M. H. Gordon.

Xmas 1900.

THE NATURAL HISTORY OF INFECTIOUS DISEASES

BY

T. W. THOMPSON

LATE MEDICAL OFFICER OF HEALTH TO THE HERTFORDSHIRE AND MIDDLESEX COMBINED SANITARY DISTRICTS

THE NATURAL HISTORY OF INFECTIOUS DISEASES [1]

In this article it is proposed to deal, mainly from the point of view of natural history, with some of the more important of that class of diseases which may at present be regarded as coming within the domain of public health.

The following is a list of the diseases about to be considered, taken in the order in which they are given by the Registrar-General :—

Measles	Mumps	Malarial Disease
Rötheln	Diphtheria	Erysipelas
Scarlet Fever	Cerebro-spinal Fever	Puerperal Fever
Typhus	Simple Continued Fever	Tuberculosis
Relapsing Fever	Enteric Fever	Croup
Influenza	Cholera	Pneumonia
Whooping Cough	Diarrhœa	Yellow Fever [2]

As regards history, it is perhaps sufficient here to say that diseases akin to those under consideration have existed from very early times. Some of the actual diseases in question are indeed clearly referred to by the earliest medical writers. Historical records also support the view that at different periods of the world's history the diseases most conspicuously affecting man have varied in type, as well as in geographical distribution from time to time.

Whatever their variations and whatever their distributions in space, it will be generally admitted that these diseases are in all cases due to the effects upon the body of poisons originally introduced from without. The precise nature of these poisons can of course only be finally ascertained by experiment, though in this matter the laboratory investigator must work in concert with the epidemiologist, each supplying the other with indications for further observation or experiment.

But it having now been demonstrated that the poisons responsible for the causation of some of the diseases in question are bound up with the life processes of microphytic organisms, not only is the view originally based upon clinical and epidemiological considerations—that the diseases of this class are due to processes allied to fermentation—completely established as regards some of them, but very strong suggestion arises that it is true also of the whole class ; indeed, so strong is the probability of such being the case that we are justified in adopting this so-called germ theory of these diseases as an hypothesis deserving to be put to the test. This course has

[1] The use of the term 'infectious' as a class name for these diseases is no doubt open to criticism, since, in the sense in which it is popularly understood, it can hardly be said to be applicable to all the maladies enumerated above. In the present state of knowledge, however, it is difficult, probably impossible, to find a class name which is at the same time applicable to all the diseases in question, and free from the objection of connoting etiological views at present unproved as regards the diseases as a class.

[2] Yellow Fever is, of course, not included in the mortality statistics of the Registrar-General.

been very generally entered upon with results which, so far, appear entirely to justify the procedure; for not only has the germ theory been found to lend itself in a remarkable degree to the explanation of the observed phenomena of disease of this sort, but the labours of the bacteriologist are day by day extending the area of demonstrated association between microphytic life processes and particular human and animal diseases. It is true that closer study of these microphytic life processes has led to some modification of the germ theory as originally formulated, in the sense of showing that, as regards an increasing number of diseases, the pathogenic influence of the microphyte upon the body is indirect, resulting rather from the action of certain chemical poisons formed during the cycle of microphytic life than from mere multiplication within the body of the microphytes themselves, as had been formerly supposed; and still further modifications of the germ theory will no doubt follow with increasing knowledge. Nevertheless it may fairly be said that the whole progress of research has tended to confirm the position of the microphyte itself as in one or other way the cause of the diseases of this class.

It is necessary, however, to state that the expression 'the cause' is here used in the popular sense only, for the *real cause* of any effect is generally, if not always, the resultant of a combination of causes. That in the present instance this point is one of practical importance, must be evident; and a moment's consideration will satisfy anyone that in the production of disease many highly important factors are operative in addition to the microphyte itself; for, although the microphyte may be truly described as an essential element in the cause, it is none the less true that for the effective operation of the microphyte certain conditions of environment are also requisite. Such conditions may be those which are external to the sufferer—e.g. climatic, topographical, or domestic; or they may be intrinsic conditions—viz., peculiarities, *quâ* susceptibility, of species, sex, age, heredity, and tissue condition of the individual.

Any attempt to unravel the problems of disease origin and propagation must take account of these several conditions, and it is to be feared that in the outburst of zeal to which the modern study of bacteriology has given rise, this consideration has been to some extent overlooked; a fact which perhaps accounts for the nature of some of the criticisms to which 'the microbe' and its supporters have been subjected by clinical physicians and students of practical medicine.

But to return to our hypothesis. The occurrence of an attack of one of these diseases presupposes the operation of microphytic life, or the products of microphytic life, upon the person attacked, and the question arises, whence came the microphyte? Was it the progeny of a similarly endowed parent, or did it arise independently of such parent?

These questions at once open up two conflicting views which have been extensively held as regards the origin of the more infectious of the diseases under consideration. On the one hand, the frequency with which such diseases have been observed to spread by infection from case to case has led to a widespread belief in infection as the only mode of their origin and dissemination. And this view—which implies that the microphyte is always derived, directly or indirectly, from a pre-existing case of disease—is still the orthodox one as regards certain well-known diseases. On the other hand, owing to the fact that in a certain proportion of instances it has been thought impossible to trace fresh attacks of a given disease to previous cases, and owing also to certain *à priori* considerations, the old theory of spontaneous generation has at different times been revived for explanation of the pro-

blem of disease origin; and it has been held by some that the microphytic causes of disease—although often, and as regards some diseases usually, descended from parent microphytes of like kind—are yet at times capable of arising independently of such parents.

As the views which we entertain with regard to these and allied questions lying at the root of the problems of disease origin and dissemination must profoundly influence our general attitude of mind towards the diseases under consideration, it is important that the subject should be considered somewhat closely. It is proposed, therefore, to inquire how far either of the two views above referred to is in accord with the facts and indications of modern science.

It will be convenient first to consider the spontaneous generation theory, or, as it is now more correctly called, the *de novo* theory.

The suggestion that one or other of these diseases has arisen *de novo* might be taken to contain the assumption that the micro-organism, either known or inferred to be the cause of such disease, has been originated afresh, presumably out of non-living matter. It becomes necessary, therefore, to ascertain whether there is any foundation for such a belief as this, and for this purpose there is occasion to refer to the general doctrine of abiogenesis.

That the origin of beings whose parental descent was not obvious should have been ascribed to spontaneous generation is not surprising, and accordingly we find that, 'the checks which experience alone can furnish being absent, the spontaneous generation of creatures quite as high as the frog in the scale of being was assumed for ages to be a fact. Here, as elsewhere, the dominant mind of Aristotle stamped its notions on the world at large. For nearly twenty centuries after him men found no difficulty in believing in cases of spontaneous generation, which would now be rejected as monstrous by the most fanatical supporter of the doctrine.' [1]

One by one, however, these beliefs have given way before better knowledge, and, as Sir Joseph Lister remarks, 'the doctrine of spontaneous or equivocal generation has been chased successively to lower and lower stations in the world of organised beings as our means of investigation have improved.' [2] These results of progressive investigation naturally incline us to regard all phases of the abiogenesis theory with grave suspicion, and to anticipate that future additions to our knowledge will still further support the view expressed in the formula *omne vivum ex vivo*. But the modern doctrine of evolution has been considered by some to put a different aspect upon the matter. It has been argued that though according to that doctrine in its wider sense the various forms of life existing upon the globe owe their existence to unbroken parental descent from the *lowest* forms of life, these lowest forms of life must themselves have arisen at some time or other, and of necessity, from non-living matter. And if once, why not again? It has been further pointed out that 'those who strictly adhere to the Evolution Hypothesis could never believe in the origination of any but the "lowest and simplest" organic forms by a process of Archebiosis. So that the gradual driving of the question back as one possibly applicable to such organisms only, is just what the Evolutionist would have expected. . . .' [3] As a purely abstract criticism upon the historical argument this at first sight appears to have weight, but when it is applied to the concrete aspect of the question it loses point. For although it may of course be true that all existing forms of life are descended from the *lowest* forms of living matter,

[1] "Spontaneous Generation." Tyndall's *Fragments of Science*, ii. 292.

[2] *Introductory Address* (University of Edinburgh), 1869, p. 12.

[3] *Evolution and the Origin of Life*, p. 29. By H. Charlton Bastian, M.D., F.R.S.

and that such again were originally evolved from non-living matter, what are the grounds for assuming that the particular forms of life, such as the bacteria, with regard to which it has been endeavoured to establish a *de novo* origin, do, as a matter of fact, constitute the lowest forms of living matter? On the contrary, may not the decided variations in function which they exhibit fairly be regarded as indicating a considerable degree of complexity of structure, notwithstanding that such complexity may not be recognisable by any means as yet at our disposal?

Furthermore, it would appear that the abstract discussion of this matter upon evolution principles by no means necessarily favours the doctrine of abiogenesis. Not only are there no sufficient grounds for supposing that we have yet found the lowest possible form of life—'the first organism' which has been assumed by some to be a necessity—but, as Mr. Herbert Spencer remarks, 'the conception of a "first organism" in anything like the current sense of the words is wholly at variance with the conception of evolution. . . . The affirmation of universal evolution is in itself the negation of an "absolute commencement" of anything. Construed in terms of evolution, every kind of being is conceived as a product of modifications wrought by insensible gradations on a pre-existing kind of being; and this holds as fully of the supposed "commencement of organic life" as of all subsequent developments of organic life. It is no more needful to suppose an "absolute commencement of organic life" or a "first organism" than it is needful to suppose an absolute commencement of social life and a first social organism. The assumption of such a necessity in this last case made by early speculators with their theories of "social contracts" and the like, is disproved by facts; and the facts, so far as they are ascertained, disprove the assumption of such a necessity in the first case.'[1]

It thus appears that the doctrine of evolution, according at least to one of its most distinguished exponents, gives no *à priori* support to the notion of the *de novo* origin of any organisms properly so called, much less of organisms such as those upon which certain diseases are known to depend—i.e. organisms capable of being identified and of propagating their kind. Of course it might be urged, as indeed it has been urged, that time like all else is relative; that an interval which to us is but a short period represents perhaps æons of time in the life history of lower organisms, and that therefore the evolution of living organisms out of non-living organic matter may be accomplished in a few hours or a few days.

However this may be, there appears sufficient experimental evidence to show that, whether or not organisms of the class we are discussing may be mentally conceivable as arising *de novo* in a short space of time, they do not as a matter of fact now so arise. And this brings us to the gist of the matter. The whole question is one which must be decided by a balance of evidence accumulated by long-continued observation and experiment. Judged by this test we have in the experiments of Pasteur, Tyndall, and others, and in the successful application of the teaching of such experiments to the practical arts, an overwhelming mass of testimony against spontaneous generation. It has been shown that, if properly sterilised and protected against accidental germ contamination, putrescible substances may be kept without alteration for indefinite periods of time; though putrefaction may be at once set up in such substances by the simple device of admitting to them a germ-charged atmosphere. All along the line, in fact, experimentation has gone to show that there is no life apart from antecedent life. Whatever, there-

[1] *Principles of Biology*, vol. i., Appendix.

fore, may have been the origin of life, the conclusion seems forced upon us alike by observation and experiment, that in the present day there is no life in the sense of anything which could with any propriety be described as a living being or organism which has not descended from antecedent life.

How far this conclusion negatives the possibility of a *de novo* origin of disease as disease will be presently seen. For it has to be noted that epidemic diseases, as we observe them, are simply the manifestations exhibited by man or other animal of the operation of certain poisons which we assume to be bound up with micro-organisms. And conditions may easily be imagined which would give rise to an apparent or even a real *de novo* origin of disease independently of any *de novo* origin of bacteria.

To summarise the point reached: We have inferred, provisionally, from what we already know of certain of these diseases, as well as from the general direction of bacteriological research, that all the diseases under consideration are due to microphytic life processes; and we have further seen no reason to doubt that the microphytes upon which these diseases depend are invariably descended from parent microphytes, and that, therefore, nothing of the nature of a true *de novo* origin occurs so far as the microphytes themselves are concerned.

Having now sufficiently cleared the ground by a review of the theory of *de novo* origin of the germs of disease, we may go on to consider the thesis which, in the absence of 'spontaneous generation' of disease germs, has been regarded by some as the only alternative—namely, the unbroken continuity of disease descent from antecedent cases. This doctrine, signifying, as it seems to do, belief that every single case of each of the diseases in question is the progeny, so to speak, of an antecedent case of the same disease, is one of little elasticity. In so far, indeed, as certain of these diseases affect the lower animals as well as man, the antecedent case of a given sample of such disease need not be a human case; but as regards others, which are held to exclusively affect man, the antecedent case must be sought in the human subject alone. But we now see difficulties in the way of this thesis, and hence arises occasion for reconsideration of the subject from the point of view which regards the microphytes upon which these diseases depend as invariably descended from parent microphytes.

Although it is no doubt true that some of the disease microphytes now in question, though capable (whether as bacteria or as spores) of existing in a latent condition outside the body for considerable periods of time, may yet be incapable of passing through their life cycle, including of course proliferation, except in human living tissue—so that diseases due to such microbes usually arise (in the present) by direct or indirect infection from a previous human case of the same disease—we nevertheless know that there are microphytes, causative of disease, which are capable of thriving and multiplying either upon human or other animal tissues; and that there are others again which have their habitat largely outside the living body, either human or animal, and are capable of thriving and multiplying upon non-living organic matter.

Accordingly, to affirm the continuity of life, from generation to generation, of the microphytic causes of disease, is by no means the same thing as to allege that human diseases descend in a continuous series from human case to human case. For instance, with respect to a particular species of pathogenic micro-organism capable of preying either upon human and other animal hosts, and of thus giving rise to a disease intercommunicable between man and other animals, it is obvious that although the micro-organisms them-

selves would, according to the view here adopted, descend in a continuous series from antecedent parent organisms, yet the cases of human disease dependent upon such organisms would not necessarily follow in a continuous series from human case to human case.

But we may go farther than this. It is evident that a given attack of disease might be due to the action of microbes which, although not developed *de novo*, had yet descended from a particular strain of microbes that had not, for a number of generations, found a habitat either in human or other animal bodies, though their remote ancestors might have done so or might not. In such an instance the disease would practically have for man a new beginning so far as this particular occurrence of it is concerned. The question therefore whether diseases do or do not descend in a continuous series from antecedent cases is one which must be worked out separately, as regards each disease, by a study both of the epidemiological behaviour of different diseases and the life history of the particular microphytes upon which such diseases depend.

So far, however, the matter has been discussed as though pathogenic property remained a constant quantity for each species of micro-organism, and throughout the life cycle of each individual micro-organism. But we are by no means assuming this either as regards the individual or the species, and many facts pointing to an opposite conclusion might be cited. It is notorious, for instance, that very considerable variations both of severity and type are observed between different epidemics of the same disease, and even between different stages of one and the same epidemic. Such variations, too, at times occur under circumstances which seem to render it improbable that they are due to corresponding variations in the conditions of the communities affected, and we thus seem led to the view that differences of severity and type are likely to be due to variations from time to time occurring in the pathogenic property of the same species of micro-organism.

And besides the variations observed in epidemics as a whole there are commonly considerable differences both of severity and type between different individual cases of a given disease during a particular epidemic; and although such individual differences are no doubt more largely due than in the former case to personal differences of the people attacked, and probably also to differences in the dose of the virus received, yet they may be in part due to differences of pathogenic property of one and the same species of microbe.

Now differences in the pathogenic property of micro-organisms of the same species may from time to time doubtless result from a variety of causes, among which temperature, moisture, light, and other factors, probably have important influence. But there would appear to be other causes, and it is to these that it seems desirable here to refer. It is well known that the pathogenic property of certain microbes varies largely according to the soil in which they are sown—accordingly, for instance, as they are cultivated in the body of one or another kind of animal. This would suggest that outside as well as within the laboratory the pathogenic property of micro-organisms that prey alike upon man and other animals may be modified from time to time by the 'soil' of the particular class of animal they chance to invade. And, further, as different species of animals seem to differ in their influence upon the pathogenic property of some micro-organisms, so possibly may different individuals of the same species differ; and certain individuals, having become infected, may be thought of as handing on the virus to others, either in a relatively attenuated or more potent form than that in which they themselves received it. Considerations of this kind may perhaps supply the key to the explanation of some of the differences ob-

served in the severity and type of different epidemics and different cases of the same epidemic. And, indeed, it is easily conceivable that the influence of the hosts upon the pathogenic function of micro-organisms may be capable of originating actually new varieties of disease.

It may be asked whether the pathogenic property of some micro-organisms may not be a function which has become superadded, so to speak, to micro-organisms originally saprophytic, as a result of their having gradually acquired ability to thrive upon animal hosts. Or, indeed, whether all pathogenic parasites may not have descended from saprophytes by a process of adaptation to environment.[1] It is so far consistent with this view that there is no sharp line of demarcation between the saprophytes and the parasites, and we should anticipate that in the struggle for existence such microphytes as were capable of adapting themselves to new environments would, in surviving, undergo variation. There would seem no reason therefore why the pathogenic function should not have been in the first instance acquired, and subsequently from time to time modified in this way. If this should be so, the evolutionary changes of micro-organisms might afford very considerable possibilities as regards disease variation, and even disease origin, altogether apart from any 'spontaneous generation' of disease germs. For, although disease germs of the more highly specialised sorts must doubtless be expected to exhibit a relatively high degree of constancy as regards pathogenic property, others, less specialised, would probably in this respect be subject to considerable variation, either in the direction of increasing capacity for production of pathogenic material, or of reversion toward innocence of function in this respect.

In view of the criticism that evolutionary changes would be expected to occupy longer periods of time than seems to be here contemplated, it has to be borne in mind that the evolution now suggested is one of function rather than a decided morphological evolution. And although, as will be pointed out presently, many micro-organisms may be remarkably stable, both pathogenically and morphologically, it has been shown under artificial conditions in the laboratory that, as a matter of fact, the functions of others may undergo considerable modification in comparatively short periods of time—and modifications so lasting as to be transmitted by heredity.[2]

The foregoing considerations serve to illustrate some of the senses in which a *de novo* origin of *disease* may still be contemplated as a possibility without any departure from the doctrine of 'no life without antecedent life,' as applied to the microphytic causes of disease.

To recapitulate. It seems that although diseases due to highly specialised obligate parasites whose life processes are dependent upon residence in a human host probably always descend from antecedent cases of the same disease, there are yet possible ways in which, independently of any 'spontaneous generation' of microbes, other diseases might, in one or another sense, be said to have a new beginning for human beings.

Illustrations, which, however, do not profess to be exhaustive of such ways, are as follows:—

1. Human disease due to a microbe capable of thriving upon the tissues

[1] See Dr. Louis Parkes' paper on 'The Relations of Saprophytic to Parasitic Micro-organisms,' *Trans. Epidem. Soc.* 1891.

[2] See Dr. Sims Woodhead's paper on 'The Relation of Modification of Function of Micro-organisms to the Virulence and Spread of Specific Infective Diseases' (*Trans. Epidem. Soc.* vol. x., N.S., 1890-91). In connection with variations in virulence of infectious diseases, Dr. Woodhead refers expressly to modification of 'function' of micro-

either of man or of other animals, might in passing from lower animals to man, seem to have a new beginning for man in that it would be untraceable to previous human disease.

2. Human disease dependent upon a microbe capable of thriving either upon man, or altogether independently of living human or animal bodies —as, for instance, on living vegetable or dead animal or vegetable matter— might also, when this microbe again attacked man after a more or less prolonged saprophytic existence, have a new beginning for man.

3. Should a microbe, already pathogenic, be capable under the influence of particular environment of still further evolving pathogenically, a new variety of disease might be expected to arise; and in this way a definite disease might arise from an indefinite malady.

4. Should a saprophytic organism be capable in certain environments of acquiring pathogenic property, an entirely new disease might arise, and in this way a new and definite disease would have *de novo* origin.

In so far, therefore, as the suggestions put forward have been valid, it would appear that the question, in perhaps its most important aspect, is not one of 'spontaneous generation' but of evolution. And while 'spontaneous generation' seems more than ever untenable, evolution in connection with epidemic diseases, or more strictly their causes, is day by day forcing itself more prominently upon our consideration. The older notion of the absolute immutability of species being now indefensible, we cannot fail to be impressed by the variations from time to time exhibited by epidemic diseases; variations so considerable as to render the classification, even at times of whole epidemics, and frequently of individual cases, an impossibility, except in a most provisional and tentative way. It would seem, too, that the fixity of type of different diseases differs in degree, a circumstance which may not unreasonably be looked upon as pointing to a gradual specialisation of the causes of disease by a process of evolution. Again, we observe that certain diseases possess points of similarity to one another, and this we are now able to regard as suggestion of descent from a common stock. All these, however, are matters rather for future investigation.[1]

In the meantime it must be pointed out that in differentiating 'diseases' from one another there is need for caution; and, looked at in the light of the germ theory and of evolution, it may be questioned whether, in some instances, differentiation has not already been carried too far. For the marking off of a disease as distinct and separate from others implies that it is due to a distinct and separate cause—i.e. from the point of view here adopted a distinct species of organism. But as regards many of the epidemic diseases it cannot for a moment be said that this is established. It has to be remembered that what we call 'a disease' is not a specific entity, but a mental conception based upon a rough average of certain morbid manifestations of bodily function and structure, frequently observed to occur in combination, and therefore assumed to be due to a separate cause. But there is obviously danger of attributing to separate causes effects which, although superficially different, may be nevertheless due to the same cause acting under different conditions. It is, for instance, held by some that this error has been committed in the case of membranous croup and diphtheria, and further knowledge may show

organisms, remarking that 'it will at once be accepted that the delicate metabolic changes in an organism are almost invariably brought into prominence long before the coarser morphological modifications can be observed.'

[1] See a paper on 'Specificity and Evolution in Disease,' by Dr. W. J. Collins. London 1884; also a further paper by Dr. Collins on the same subject in *Public Health*, October 1889.

it to have occurred with regard to various other diseases, as, for instance, certain forms of diarrhœa and enteric fever. Indeed, we have yet to learn that some of the diseases which, though alike in kind, differ in malignity, and which are now regarded as due to different species of organisms, are not in reality due to different biological phases of organisms of the same species. In building up an opinion as to the separate nature of any disease, not only its clinical features, but all ascertained facts relating to its natural history, must be fully taken into account. Its period of incubation, duration, period of infectiveness, complications, sequelæ, conditions of occurrence, modes of dissemination, relations (of coexistence, &c.) to other diseases, and particularly its age and seasonal incidence and geographical distribution, must be carefully compared with similar facts as to other maladies.

But a caution of another character must be offered. If it is necessary to abandon the notion of the *absolute* immutability of diseases, it is of the utmost importance to avoid falling into the opposite error of underestimating the relative fixity of type which some of them have evidently acquired. And there is nothing whatever in the suggestions put forward as to the possibility of variations in existing types of disease occurring as a result of variation of function of their microphytic causes, or of the possibility of entirely new diseases arising from the evolution of saprophytic into parasitic bacteria, in the least degree justifying the conclusion that all existing epidemic diseases may have a present day origin of some such kind. Certain diseases may, as previously pointed out, and doubtless do, depend upon highly specialised obligate parasites, incapable now of thriving except in the tissues of human hosts. Such organisms, though themselves a product of evolution, may have been evolved in the remote past under conditions of environment never since, and perhaps never again to be, reproduced.

Notwithstanding, therefore, that all pathogenic micro-organisms are doubtless—especially as regards their pathogenic function—subject, from time to time, to minor variations within the limits of the species, yet many species of such organisms may have long since attained a high degree of fixity of type, and thus exhibit now but little tendency to variation beyond such limits. That such is actually the case with regard to the microphytic causes of some of the diseases under consideration there seems ample epidemiological evidence to show.

In this connection it is important to note that occasional difficulty in tracing particular attacks of a given disease to previous cases is far from justifying hasty conclusion that such attacks have had an independent origin. The probability of such being the case must depend upon a comprehensive view of the ascertained facts with regard to the disease in question—at least, that is, pending definite knowledge of the life history of the microphyte upon which the disease depends. The multifarious ways in which infection may have been carried, and the difficulty of tracing its carriage by many such ways, must be fully taken into account. Allowance must also be made for errors of diagnosis, untreated, and concealed cases, which largely add to the difficulty of following out the causal association between successive and connected attacks. If this difficulty is met with in the early cases of an outbreak, the possibility of tracing back the outbreak to some previous prevalence in a more or less remote neighbourhood, to which it may in reality have been due, is entirely prevented.

Moreover, the presumption that a disease never in the present day arises except as a result of infection, or inoculation, from a previous case of the same disease, will certainly be strengthened if it should be ascertained that the disease in question did not occur in certain districts until introduced from

without; or, if having once been prevalent in such districts, it should have entirely disappeared after measures for the prevention of its spread by infection or inoculation, and for the prevention also of its reimportation, had been taken. Measles and rabies may be mentioned as instances in which these two arguments respectively must be allowed as having considerable weight.

Having now considered some of the aspects of the problem of disease origin, certain other general matters connected with the class of diseases under consideration must be discussed.

Diseases may be Epidemic, Pandemic, or Endemic.—As regards their more general manifestation the diseases now being dealt with are usually described as either epidemic, pandemic, or endemic.

The terms 'epidemic' and 'pandemic' respectively signify the tendency of diseases to spread more or less rapidly in a given community or over a vast area of the world's surface, in either case with a large indifference to local circumstances. In this aspect, therefore, the distinction between epidemic and pandemic disease is one of degree. The term 'endemic' signifies that a disease tends specially to abide among the inhabitants of a particular locality, and is therefore presumably largely governed by local conditions.

In the present day these distinctions are being somewhat reduced in importance, since study of the geographical distribution of disease appears to show that epidemic, and even perhaps pandemic, diseases have a preference for certain areas; while, on the other hand, increasing knowledge of so-called endemic diseases is giving greater prominence to the epidemic character of some of them, as, for instance, cholera. In so far, however, as diseases do display one or other of the characters mentioned, the probable explanation would seem to be that epidemic and pandemic diseases are due to microbes which especially thrive and multiply in living animal tissues, whereas endemic diseases are for the most part due to microbes whose habitat is more largely outside human and animal bodies, and therefore more influenced by local circumstances.

Diseases may spread by Infection, Contagion, or Inoculation.—With reference to the manner in which they are communicated from one individual to another, these diseases are also frequently spoken of as infectious, contagious, or inoculable. The three terms equally imply the transmission of infective material from one person or animal suffering from a given disease to some other person or animal in whom the disease in question becomes thereby established. But they also are intended to indicate distinctions as to the methods by which such transmission is effected, and the means by which the poison gains access to the system of the recipient. In the case of *inoculation* the poison is conveyed, either directly by actual contact with the diseased body, or indirectly by the agency of some surgical instrument or other article, from the person or animal affected to the person or animal previously unaffected, and gains access to the system of the latter through some breach of surface in the skin or mucous membrane. *Contagion*, on the other hand, means transmission of the poison by actual contact, but without any initial breach of surface in the recipient; while *infection* refers to conveyance of the poison in a more indirect way through the medium of air, water, soil, food, clothing, letters, &c., and its entrance to the system of the recipient through one of the mucous tracts, or possibly even through the skin, but in either case, again, without any decided breach

of surface. These terms are useful as broadly indicating different modes by which disease transmission may occur, but care must be exercised in applying them as exclusive modes of dissemination to the different diseases, or in basing disease classifications upon them. Certain infections are, so far as we know, only capable of being transmitted by inoculation, but others, in which inoculation is the normal method of communication, are said to be also capable of being inhaled. Further, some diseases are both infectious and inoculable.

In the present day the tendency is to discourage the use of the term 'contagious,' but if kept within proper limits, it should still have a legitimate application, and there appear no sufficient grounds for banishing it from medical literature. There is a real distinction between the transmission of a disease by actual contact, which is direct, and transmission by air or fomites, which is indirect, and contact certainly plays a larger share in the spread of some diseases than others.

Certain Diseases have special Seats of Invasion.—Certain diseases appear to have special seats or points of invasion, that is to say, the viruses upon which they depend usually attack the body by some special channel or channels, and possess little if any ability of primarily establishing themselves elsewhere. This appears to be the case with regard to the poisons of diarrhœa, cholera, and enteric fever, which make their assault upon the intestinal mucous membrane, and with regard to the poison of pneumonia, which attacks the lungs.

These facts, as Flügge points out, are of considerable importance with respect to the spread of such diseases, for it is not sufficient, in cases of the kind, that the poison should simply be brought to the body, but it must be conveyed also to the particular part of the body which is vulnerable to it. Here we find an analogy in the behaviour of the parasitic fungi which produce the diseases of plants. Some of these attack the flowers, some the fruit, and some the roots.

As regards other pathogenic micro-organisms, such as those causative of anthrax, tuberculosis, &c., the points of invasion are more numerous.

Epidemic Diseases have a Period of Incubation.—When a person has received the poison of one of these diseases a period elapses before definite and recognisable symptoms of the action of the poison manifest themselves. This interval, which is described as the period of incubation, varies considerably as regards different infections, ranging from a few hours in the case of some of them, as perhaps erysipelas, diphtheria, and scarlet fever, to weeks in the case of others, as syphilis and rabies, and even probably years in the case of leprosy. For each different infection, however, the period is comparatively constant; though here, again, variation, within certain limits, occurs in different individual cases of the same infection, and the period is more constant with some infections than others.

It is only by an accurate knowledge of the periods of incubation in the different diseases that we can be enabled to say when a person who has been exposed to a given infection may safely be regarded as having escaped attack, and can therefore be looked upon as, presumably, free from danger to others. Great care, too, is required in studying the periods of incubation in order that fallacies due to multiple or sustained exposure may be eliminated. 'The only cases in which it [a period of incubation] can be positively determined are of course those in which there has been but a single exposure to contagion; but others, in which the exposure (though repeated) began only a few days before the patient's illness showed itself, are valid as

proofs of short incubation; and yet others in which the exposure ceased many days before he felt ill, are valid as proofs of long incubation.' [1]

No detailed explanation of the incubation period can at present be given. It is often broadly stated to be the period occupied by the multiplication of the poison. This, however, if intended to signify the multiplication of micro-organisms, is a very unsatisfactory explanation when we remember, as already stated, that the incubation period extends over weeks in some diseases. Mere multiplication of micro-organisms can hardly be regarded as requiring such lengthened periods of time, and there can be little doubt that some far more complicated process has to be gone through prior to the general infection of the individual. What this process is remains for future study, and it has to be remembered that the process may possibly be a very different one for different diseases.

The following suggestions may be put forward as perhaps worth consideration.

1. If it is a fact that bacteria are incapable of directly penetrating healthy mucous membrane, as Flügge seems to affirm, some little time would doubtless be required for bringing about the local changes at the seat of lodgment, which are necessary for the entrance of the bacteria.

2. Certain diseases at least appear, as already said, to be due to the action of chemical poisons produced by the micro-organisms causative of them, rather than to the simple presence of the organisms themselves. As regards such diseases, a time—and a time which may be different for different infections—is perhaps required for the accumulation of a sufficient quantity of the poison to produce the several phenomena of the disease.

3. The excessively long incubation periods, as in rabies and leprosy, would seem to suggest either some peculiarity in the life history of the micro-organisms upon which such diseases depend, or perhaps the necessity for some gradually effected change in the tissue condition of the recipient prior to the full operation of the pathogenic properties of the micro-organisms. Or, again, it may be that the particular poison locally produced is also locally stored up, and later on discharged from the local tissues and disseminated throughout the system.

Protection.—A very important phenomenon in the natural history of these diseases is that certain of them confer protection or immunity against future attacks upon persons who have once suffered from them. The degree of protection thus imparted varies considerably, however, in different diseases, and in different individuals suffering from the same disease. It also differs, too, according to the age of the individual at the time of primary attack. As regards some diseases it is usually stated that the protection is of life-long duration. This doubtless often is so; but here it becomes interesting to ask how far the life-long protection, which apparently results from a single attack of one of these diseases, may be due to occasional subsequent exposure of the individual to the same infection, and consequent renewal of protection. It is conceivable, for instance, that a person may derive absolute protection for a certain period. Towards the close of this period he may be again exposed to the same infection, but being still partially protected by the antecedent attack, his re-infection is not followed by conspicuous manifestation of the disease. He may suffer perhaps from a mere undefined malaise which escapes serious attention, but nevertheless serves to renew his protection. In this manner the protection of such diseases as measles and scarlatina may perhaps be renewed from time to time. It is by no means

[1] *The Principles and Practice of Medicine.* C. Hilton Fagge, M.D., F.R.C.P. Edited and completed by P. H. Pye-Smith, M.D., F.R.S. 2nd edit. i. 27.

uncommon to find, for instance, persons in infected households, who have previously suffered from scarlet fever, again exhibiting sore throat and malaise. This doubtless means a re-infection, and renewal of protection; and it becomes an important question how far such persons, while passing through these later mild attacks, are capable of infecting others. In re-vaccination we have experimental confirmation of the renewal of protection.

Another interesting question is whether any degree of protection is capable of being transmitted by heredity. The exceptional virulence of measles when implanted upon virgin soil, as in the case of the Fiji outbreak, suggests the possibility of this being the case, notwithstanding that the mortality of that outbreak was doubtless largely due to a neglect, based upon ignorance and superstition, of all necessary precaution.

The protection afforded by some of these diseases is, however, far less than that afforded by others. Of certain of them, indeed, it is said that they do not protect at all, and that some, as erysipelas, even predispose to future attacks. On the other hand, it has been suggested that all microbic diseases protect for a certain period, though in some instances only a short one; that otherwise it would be difficult to understand such diseases ending except in death, for it has been thought that in the absence of any protection afforded by the disease process a person once infected would keep his disease going perpetually by auto-infection. However this may be, there can be no question that for many people at least, the protection, if any, afforded by an attack of some of these diseases is exceedingly slight, and the question might be raised, as for instance in the case of erysipelas, how far this is due to the disease itself, and how far to peculiarity of tissue of persons especially subject to it. The problem is admittedly a difficult one, and time and observation are wanted for its solution; moreover, it is complicated by the apparent ability of certain infections to lie dormant in individuals, and to recrudesce in them at uncertain intervals. As to this, there seem no *à priori* reasons why microbes should not remain in the system, held in check by the conditions which have brought their obvious manifestations to an end, and consequently conferred a temporary protection on their host. Ultimately the forces may become reversed, and the microbes gain a second ascendency, to be subsequently in similar manner reduced to temporary quiescence. A thesis of this sort would seem necessary for explanation of the recurrent attacks of ague from which persons frequently suffer years after removal from malarious districts.

Original liability to attack by epidemic disease varies in different individuals, some appearing by nature almost immune, while others exhibit a marked degree of susceptibility. Such contrasts, there can be little doubt, are due to differences—sometimes hereditary and sometimes acquired (in other ways than by attack)—in the tissue condition of the several individuals —and especially perhaps in the condition of the tissues (mucous membrane, &c.) at the points of invasion special to the different diseases. The marked tendency to phthisis observed in certain families probably affords an instance of transmitted liability to infection, while the well-known predisposing influence of unwholesome conditions of life, overcrowding, starvation, &c., illustrates acquired increase of susceptibility to certain infections. In this connection it is worthy of note that the general experience of epidemiologists to the effect that conditions tending to lower vitality predispose to attack by certain diseases, seems recently to have received indirect experimental support.

Susceptibility to certain infections is also no doubt increased by other conditions, such as cold and damp.

Influence of Age and Sex upon Original Liability to Attack.—The age and

sex incidence of the different diseases, as judged mainly by mortality statistics, will be given in the appropriate sections. Speaking generally, it may be said that, as regards certain diseases which mainly affect the young, the age incidence observed is no doubt in part due to the fact that since so large a number of the individuals of a community are attacked during childhood, the protection thus acquired diminishes the number of adults susceptible to attack. This, however, cannot be regarded as the sole factor, but it probably serves to exaggerate, or perhaps modify, the true age incidence, which there can be little doubt is of a deeper and more fundamental character. Some diseases for instance, on the contrary, appear to pick out adolescents or adults in preference to children, while some, again, of those diseases which especially affect children, as scarlatina, rarely attack very young infants.

Sex incidence in adults is no doubt in part regulated by occupation—that is to say, according to the different occupations of the two sexes, so generally is their chance of exposure to certain infections. At the same time it is now tolerably certain that, independently of occupation, the actual susceptibility of the two sexes varies as regards some diseases.

Influence of Season upon the spread of Epidemic Diseases.—It is well established that some of the diseases under consideration exhibit a decided preference for certain seasons of the year. These seasonal variations are no doubt largely due to climatic influences upon the life phases of the micro-organisms upon which the different diseases depend; that is to say, each species of micro-organism probably has its particular season of the year in which the meteorological conditions most favourable to its development, at all events as regards pathogenic property, are at a maximum. Seasonal variations may, however, be partly, and in the case of some diseases largely, due to the effects of season on the recipients—either the direct effects, as influencing susceptibility, or the indirect effects as modifying the habits of life in such a manner as to facilitate the process of infection from case to case.

Periodicity in Epidemic Diseases.—In addition to the seasonal variations these diseases prevail more extensively in some years than others. This, of course, may be explained, and in part correctly explained, in the same manner as seasonal variations; that is to say, it may be concluded that unusual prevalence is partly, and sometimes perhaps entirely, due to climatic and other conditions which are unusually favourable to the development and dissemination of pathogenic micro-organisms. But as regards the varying prevalence of some of these diseases at least, it is becoming increasingly evident that there is a more or less regular periodicity—a cyclical character, in fact.

This subject was dealt with by Dr. Arthur Ransome,[1] and has again, quite recently, been studied by Dr. B. A. Whitelegge.

Dr. Ransome pointed out that a study of 'the track taken by any of the more common and fatal of the infectious diseases throughout a long series of years' showed them to 'observe definite periodic times or cycles,' which he described as a succession of waves, the periods covered by the waves differing for different diseases. He also showed that as regards some maladies, 'such as scarlet fever, whooping cough, and perhaps also small-pox,' if a sufficient number of years be taken in review, which is rendered possible by the Swedish mortality statistics, there is indication of 'certain larger as well as smaller disease waves.'

Dr. Whitelegge corroborates Dr. Ransome as regards the occurrence of

[1] *Proc. Lit. and Phil. Soc.*, Manchester, Jan. 27, 1880; and *Trans. Epidem. Soc. Lond.*, vol. i., N.S., 1881-82.

the larger and smaller cycles, and by a further study of the facts concerning them, as well as by an extension of his survey to other disease periodicities, he is led to formulate certain general propositions. Disease undulations or waves, he considers, are of two essentially different kinds—the accidental, or, more correctly, 'superadded' waves, and the fundamental or true cycles.

The characteristic of the 'superadded wave' is that it is not attended by any regular and progressive increase, and subsequent decrease, of virulence. It is a wave of mere prevalence, and is probably but a reflex of changes in the environment. The following Dr. Whitelegge regards as instances of 'superadded waves:'—

(*a*) The weekly wave, which he has shown to occur at Nottingham with respect to scarlet fever. This he considers due to the diminished opportunity on the Sunday for the spread of the disease by school attendance.

(*b*) The annual seasonal wave, as regards which he finds that the rise in the number of cases does not carry with it a proportionate rise in the number of deaths, nor is the fall in the number of cases attended with an equivalent fall in the number of deaths.

(*c*) Epidemics caused by specifically contaminated milk or water, inasmuch as they are due to artificially increased opportunities for the spread of infection.

The true fundamental cycle, on the other hand, is characterised by an increase both of prevalence and severity.[1] A typical instance is found in the long cycle extending over a considerable number of years. Study of such cycles shows them to embrace, and indeed to be made up of, a number of the 'short' cycles (quinquennial, &c., according to the particular disease), each of which short cycles displays an increase both of prevalence and severity, as compared with its predecessor, until the maximum of the 'long' cycle is attained. Then follows a decrease both in the prevalence and severity of each successive short cycle. But in regard to scarlet fever, at all events, these shorter five- or six-year cycles, which make up the larger wave, are themselves demonstrably truly cyclic in character. They are waves of increasing, followed by decreasing, severity. Whether the same can be affirmed of the biennial rhythms of measles seen in certain towns is doubtful, the available statistics being insufficient to determine the point.

The 'true' cycles might conceivably be due to corresponding cyclical changes in environment, such as meteorological changes; but Dr. Whitelegge considers it likely that they are of a more fundamental character, probably associated with microphytic evolutionary processes.

MEASLES

Synon.: *Morbilli*; *Rubeola*. Fr. *Rougeole*; Ger. *Masern*; It. *Rosolia*.

History and Geographical Distribution.—There can be little doubt that measles is a disease of ancient origin, though for many generations it was confounded with other maladies, notably small-pox and scarlatina. With small-pox it continued to be confounded until the sixteenth century, and with scarlet fever till the days of Sydenham. Nevertheless it is apparently referred to as 'hasbah' in the writings of the Arabian physicians, who

[1] The increase in severity being indicated by a rise in the case mortality, and by the tendency of the disease to attack people at ages usually more or less spared by it as well as persons protected by previous attack. See Milroy Lectures delivered since the above was written. *Lancet*, February 25, and March 4, 11, and 18, 1893.

regarded it (along with scarlet fever, no doubt) as a variety of small-pox. During the Middle Ages the disease would seem to have been widely prevalent in Europe and Asia, generally, according to the accounts, in association with small-pox.

The original seat or native home of measles is unknown, but nowadays the disease has a world-wide distribution, and, now as formerly, in localities not prone to it, its presence might doubtless always be traced to introduction from without. According to Hirsch,[1] it has been four times introduced into Iceland (1664, 1694, 1846, and 1868), and four times into the Faröe Islands (1781, 1846, 1862, and 1875), those localities remaining free from it during the intervals; it reached the western hemisphere 'soon after the arrival of the first European settlers;' it appeared in the Hawaiian Islands in 1848; in the Australian continent, Tasmania, and New Zealand in 1854. There appear to be no grounds for supposing that it had existed in these countries prior to its importation from without, and it would thus seem probable that its general distribution throughout the world had been brought about by human travel and colonisation.

Measles is now well established, occurring in frequent epidemics, throughout most of Europe, Asia, America, and those parts of Africa of which we have definite information on the subject.

In England it is everywhere from time to time prevalent.

During the decennium 1871–80 the registration counties in which the mortality from measles among children under five years of age was above the general average were, beginning with the highest, Lancashire, Devonshire, Monmouthshire, London, Cumberland, Leicestershire, and the West Riding.[2] It is interesting to note that the high measles mortality was not confined to densely populated counties, and, therefore, cannot have depended entirely upon the increased facilities for spread which aggregation affords. It also appears that the geographical distribution of measles in this country differs considerably from that of diphtheria, and to some extent from that of scarlet fever, though, as regards the latter, the question is complicated by hospital isolation, which has not operated in the case of measles.

Periodicity.—At different times measles has tended to become widely prevalent over large tracts of country, assuming almost a pandemic character. No regularity in the times of appearance of these major epidemics seems to have been made out; and as regards the lesser epidemics of a more local character, the inter-epidemic interval varies greatly. Thus the intervals between outbreaks have been variously stated as from two to six years for different places and by different observers. In view of these discrepancies, and also of the different length of interval between epidemics of measles in particular places, Hirsch disbelieves in any definite periodicity. Without drawing the line too fine, however, it may be said that in large communities the disease tends to occur epidemically at intervals of from two to four years, disappearing more completely between these epidemic visitations than is the case with scarlatina and some other diseases; and that in small communities, especially among the populations of rural districts, the intervals are less regular, and longer.

The Influence of Climate.—Upon a cursory survey of the facts it would at first sight appear that measles is uninfluenced by climate; Hirsch, indeed, in view of the practically universal distribution of the disease throughout

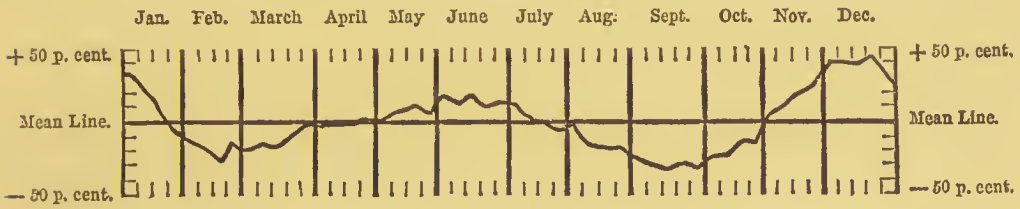
[1] *Handbook of Geographical and Historical Pathology*, 1883, i. 156, by Dr. August Hirsch. Translated by Charles Creighton, M.D. New Sydenham Society.

[2] Forty-seventh Annual Report of the Registrar-General, p. xiv.

the world, concludes that its occurrence 'is quite independent of *climatic influences*.' It is doubtful, however, whether such an inference can be legitimately drawn from the universality of its distribution, which may simply mean that the influence of climate is masked by the high infective power of the disease and other conditions, but some climates may nevertheless be more favourable to its occurrence than others. That climate is not without influence on measles would seem indicated by the consideration, which Hirsch himself points out, that 'wherever it (measles) has occurred, the influence of *certain kinds* of weather, depending on the *seasons*, has been observed to have a marked effect upon the frequency of its outbreak and the extent of its prevalence.'

And of Season.—This influence of season appears to have been everywhere observed, and, taken generally, the warmer periods of the year have been found least to favour the prevalence of measles. In temperate zones, of 530 epidemics of measles in Europe and North America which Hirsch records, 339 occurred in the colder, against 191 in the warmer months. And the same thing has been observed in the tropics. In this country the effect of season upon urban measles has been studied by Dr. Buchan and Sir Arthur Mitchell, and by the Registrar-General. The former, whose conclusions are based upon the mortality statistics of London for the thirty years 1845–1874, as recorded in the weekly returns of the Registrar-General, give the following diagram[1] as illustrating the weekly variations in the London mortality of measles.

Measles, for all Ages and both Sexes.



In reference to this diagram they make the following remarks: 'The measles curve is remarkable in showing a double maximum and minimum during the year, the larger maximum occurring in November, December, and January, and the smaller in May and June; the larger minimum in August, September, and October, and the smaller in February and March. The most rapid fluctuation takes place in the fall from Christmas to the middle of February, the weekly deaths falling from forty-two to twenty-one. This is one of the steadiest curves from year to year, both maxima being well marked in nearly every one of the thirty years in December and June respectively, and the minima also being well marked.'

It must of course be remembered that the curves under consideration are based on mortality statistics, and that for indication of the development of the measles to which the data refer, the curves should be moved some three or four weeks back throughout the year, to allow for the period of incubation and the period of illness prior to death.

Mortality.—Apart from sex and age influences, which will be dealt with

[1] We are indebted to the courtesy of Dr. Buchan and Sir Arthur Mitchell for permission to reproduce this and other diagrams from their paper on 'The Influence of Weather on Mortality' (*Jour. Scottish Meteor. Soc.*, July 1874–July 1875). The Registrar-General in his Annual Summary for 1890 has published a more recent seasonal mortality curve for measles, based upon the deaths from measles in London during the fifty years 1841–1890. The Registrar-General's curve for the longer period, however, bears a very close resemblance to that given above.

later, measles mortality must be considered from two points of view, e.g. the actual mortality or total deaths, and the case mortality (fatality) or proportion of deaths to attacks. The actual mortality, moreover, to be intelligible, must be reduced to some standard of measurement, as, e.g. the number of deaths per thousand or million of the population. The table below shows the measles mortality in England and Wales for each year from 1838 to 1890, with the exception of the four years (1843–46) for which the causes of death were not extracted by the Registrar-General.

It will be seen that in this country measles is responsible for the deaths of from 6,000 to 14,000 persons each year. It will also be seen that there has been a decided rise in the mortality from measles during the last ten years. How far this is the result of any special advantage measles has of late years gained over us, or how far it is merely the expression of a cyclical character of the disease, is an interesting problem.

Fatality.—The case mortality of measles is capable of varying within very wide limits. By some observers it is stated to average 5, by others 10, per cent. of attacks, while there is plenty of evidence that the fatality of this disease may in a given epidemic range as low as 2 per cent., and in another epidemic be as high as 40 or 50 per cent. Probably there are two main factors in inducing high fatality, the one belonging to measles itself—extra intensity of infection—the other bound up with unfavourable conditions—overcrowding, bad and deficient food, fatigue—of the population invaded; and of course, where these factors are concurrent, the death toll must needs be high. In this way may be explained the high measles fatality from time to time observed among troops on active service, and among prisoners of war. Thus

TABLE I

Year	England and Wales		Average annual death-rate per million living for each quinquennial period	Year	England and Wales		Average annual death-rate per million living for each quinquennial period
	Total deaths	Death-rate per million living			Total deaths	Death-rate per million living	
1838	6,514	426		1865	8,562	405	457
1839	10,937	705		1866	10,940	511	
1840	9,326	591	539	1867	6,588	304	
1841	6,894	433		1868	11,630	528	428
1842	8,742	542		1869	10,309	464	
1843	No data; causes of death not extracted by Registrar-General			1870	7,543	335	
1844				1871	9,293	408	
1845				1872	8,530	368	
1846				1873	7,403	316	373
1847	8,690	507		1874	12,255	517	
1848	6,867	395	403	1875	6,173	257	
1849	5,458	311		1876	9,971	408	
1850	7,080	398		1877	9,045	366	
1851	9,370	521		1878	7,765	310	385
1852	5,846	320		1879	9,185	362	
1853	4,895	266	399	1880	12,328	478	
1854	9,277	498		1881	7,300	280	
1855	7,354	391		1882	12,711	483	
1856	7,124	373		1883	9,329	350	413
1857	5,969	310		1884	11,324	419	
1858	9,271	476	425	1885	14,495	533	
1859	9,548	485		1886	12,013	436	
1860	9,557	479		1887	16,765	602	
1861	9,055	450		1888	9,784	347	468
1862	9,800	481	457	1889	14,732	518	
1863	11,349	550		1890	12,386	439	
1864	8,323	397		1891	12,673	436	

in the American Civil War, a mortality of 20 per cent. of those attacked by measles is said to have occurred in two large hospitals. 'In Paris during the siege (January 1871), out of 215 of the Garde Mobile who took measles, 86, or 40 per cent., died; and the mortality reached very nearly the same figure among the French troops who returned to Paris after the Italian War, 40 out of 125 cases dying in one hospital (whose sanitary condition was bad) with severe intestinal symptoms.'[1] Again, according to Masterman, 'at the beginning of the Brazilio-Paraguayan War, an epidemic of measles swept off nearly a fifth of the National Army in three months, not from the severity of the disease, for I treated about fifty cases in private practice without losing one, but from want of shelter and proper food.' So, too, among many uncivilised communities the mortality from measles imported for the first time has been excessive, in some cases whole tribes having been swept away. As instances may be mentioned the outbreaks on the banks of the Amazon, at Hudson's Bay, the Cape, Tasmania, Mauritius, and the Fiji Islands. The excessive mortality in such outbreaks has often been attributed to the fact that the sufferers in question presented a virgin soil to a very intense virus. This may have been, and probably was, one of the factors which contributed to the result; but from official and other reports it appears that important factors were neglect, superstitious practices, and unwholesome conditions of life. Among the small proportion of persons who in some of these outbreaks were treated in hospitals, and were thus more favourably circumstanced than the majority, the mortality was not extravagant. Referring to the terribly fatal epidemic at Fiji, Dr. Squire says: 'The excessive mortality resulted from terror at the mysterious seizure and the want of the commonest aids during illness.' Numbers of the people, it appears, either kept closely shut up in their unventilated houses, or rushed into the streams during the height of the illness. This epidemic carried off from one-fifth to one-fourth of the entire population, but of 143 native constables who were under the treatment of Dr. Cruikshank, only nine deaths were reported, 'most of these resulting from evasion of needful precautions.'[2]

Influence of Race.—There is not sufficient evidence as to how far difference of race, as such, influences liability to attack by measles or mortality from that disease. Neither can anything definite as yet be said as to the influence of sex and age so far as liability to attack is concerned. But both sex and age appear to influence mortality, and therefore probably also liability to attack.

Of Sex.—Speaking for England and Wales, the rate of mortality among children under two years of age is greater for males than females. At all age-periods above two years it is greater among females. Owing to the fact of the main incidence of the disease falling upon the early years of life, the mortality is, on the whole, somewhat greater among males than females.[3]

Of Age.—As to age in this country, about 98 per cent. of all deaths from measles occur among children under ten years of age, 90 per cent. among those under five, 75 per cent. among those under three, and 60 per cent. among those under two, the maximum mortality as well as the maximum rate of mortality being in the second year of life. This very marked incidence of measles upon the young is no doubt largely owing to the frequent epidemicity of the disease, which results, at any rate as regards towns, in a very large proportion of persons being attacked during childhood, either to die then or survive protected against further attack. For where measles

[1] Hirsch, *op. cit.*, i. 168.

[2] Paper read before the Epidemiological Society by Dr. Squire, *Med. Times and Gaz.*, 1877, p. 323.

[3] Fifty-first Annual Report of the Registrar-General, p. xxiii.

has been introduced among unprotected communities, as in the case of the Faröe Islands, it can attack most persons brought in contact with it, irrespective of age. Even in this country, adults who have hitherto escaped the disease are often seen to take it readily if exposed to the infection, and some of them suffer from it severely. Notwithstanding all this, however, it is extremely likely that measles, although capable of freely attacking unprotected persons of any age, yet has an affinity for the early years of life. And there seems no doubt at all that the disease is more dangerous in delicate children and in infants under four or five years of age (excluding the first six months) than in the latter stages of childhood.

Cause and Mode of Dissemination.—Measles is unquestionably due to a poison which is capable of being transmitted from the sick to the healthy, and of multiplying in the system of the recipient. Hence the probability of this poison being a micro-organism, though no such organism has at present been demonstrated to stand in a causal relation to the disease.

Whatever may have been the remote origin of the disease, there is no evidence that it now ever occurs except as a result of direct or indirect infection from a previous human case, and all that has been said with regard to its general history points to this as the main, if not the only, mode of its occurrence in modern times. In view, however, of recent evidence in the case of some other diseases, the possibility of measles affecting the lower animals as well as man, and being communicable from them to him, must not be lost sight of.

The poison of measles is held to be given off by the breath and mucus—possibly also by desquamating cuticle, though this is less certain. The recipient is no doubt, as a rule, infected through the respiratory tract. Generally the disease is transmitted directly from case to case; but the poison is probably capable of being air-borne, especially in ill-ventilated rooms, to a greater extent than has often been supposed. The poison also can cling to surfaces, and so may be carried by fomites. There is at present no evidence of its being conveyed by water, milk, or food.

The infection is freely given off during the early catarrhal stage of the disease, before the eruption appears; throughout the illness, and to some extent during convalescence. The periods of greatest infectiousness, however, are during the pre-eruptive stage, and while the rash is present. This early infectiousness of measles is a very potent factor in the dissemination of the disease, for during its pre-eruptive stage, children, though in a highly infectious condition, are commonly allowed to mix with others under the impression that they are simply suffering from common colds. In this way there can be no doubt that schools, churches, and other places of public resort play an important part in the spread of measles.

Periods of Incubation and Infectiveness.—In the naturally acquired disease the usual period of incubation is probably about eleven days, though there is not uncommonly a variation of several days on either side of this. Panum, in the Faröe Islands, found that the *rash* usually appeared on the fourteenth day after a single exposure. Goodhart[1] says that, 'though liable to modification within limits of three or four days either way, the incubation period centres round ten days.' According to Finlayson,[2] who has collated the opinions of recent authorities on this and allied matters, Murchison stated the incubation period as ten to eleven days; Vacher as three to seventeen, usually ten; Squire as eight to eighteen, usually eight to twelve;

[1] J. F. Goodhart, M.D., F.R.C.P., *Diseases of Children*, 2nd edit., p. 128.

[2] James Finlayson, M.D., the *Glasgow Med. Jour.*, May 1889.

Richardson ten to fourteen; Stephenson fourteen days; Clement Dukes ten to fourteen, usually eleven; Newsholme ten to fourteen, usually twelve to fourteen.

As has been said, the infective period, i.e. during which the patient is capable of infecting others, must be considered as at all events beginning with the very earliest symptoms, some days before the rash appears, and it perhaps extends through most or all of the incubation period. The subsequent duration is variously stated by different observers as somewhere between two and four weeks from the date of appearance of the rash, though it is generally agreed that infection is usually over by the end of the fourth week, provided all cough and desquamation have ceased.

Protection.—One attack of measles usually confers a lasting protection against future attack. This was illustrated in the outbreak of 1846 in the Faröe Islands, under circumstances which gave to the experience the conclusiveness of a carefully planned experiment. The disease had been unknown in the islands since 1781, a period of sixty-five years. During the six months following its reimportation in 1846, according to Panum,[1] no less than 6,000 persons, out of a total population of 7,782, contracted the disease. But every one of the old persons who had been attacked during the previous visitation escaped in 1846. Further, that their immunity was not simply due to age was evident from the fact that the other old persons who had been alive in 1781, but had then escaped, now took the disease, practically without exception. Second attacks, however, do sometimes occur. Occasionally also relapses are met with.

RÖTHELN

Synon.: *German Measles, False Measles, Epidemic Roseola, Rubeola, Rubeola sine Catarrho, Rubella*; Fr. *Rougeole.*

About the middle of the eighteenth century—that is, shortly after the complete separation of scarlet fever from measles—mention began to be made in medical literature, under the titles of 'roseola' in England and France, and 'rubeola' in Germany, of a malady seemingly different from scarlet fever and from measles, but having some of the characters of both.[2] From that time to the present, outbreaks have now and again been met with which, from a clinical view, it has similarly been found difficult, or impossible, to regard as either true scarlet fever or measles, and accordingly a belief in the existence of a third exanthem of this kind has gradually gained ground, until at present it is very generally held.

Many have regarded this complaint as a hybrid of scarlet fever and measles, but the view most generally adopted by modern authorities is that, although superficially resembling both scarlet fever and measles, it is an entirely distinct and specific disease.

Apart from its special clinical features, for which the reader must be referred to works on general medicine, this disease is held by such authori-

[1] *Archives Générales de Médecine*, April 1851.

[2] According to Prof. Thomas (Ziemssen's *Cyclopedia*), some affirm that an ailment of this kind was referred to by the Arabian physicians as 'hhamikah.' Prof. Thomas also refers to the possibility of the 'benignant "Rossalia epidemies" of earlier centuries' having been rötheln. But this, he says, cannot be ascertained owing to lack of accurate descriptions.

ties to occur in epidemics, to cause little or no mortality, to be usually unattended by sequelæ, to protect against itself, but not against either scarlet fever or measles, 'nor do attacks of either of these diseases in any way modify the liability to this one.'[1] It is said to spread by infection, but the infection is stated to be 'apparently less active than that of measles, and less persistent than that of scarlet fever, for more escape in a house or school during an epidemic of rubeola than during one of measles, and cases seldom occur after an interval of cessation.'[2] Its period of incubation seems to be somewhere about a fortnight; and the period of infectiveness from two to three weeks. According to Dr. Squire, rötheln is, like measles, 'contagious even before the rash is thrown out. . . .' In this country, or at least in London, it is apparently most common from March to June.[3] In an epidemic of what was apparently rötheln at Calcutta, recorded by Surgeon-Major McLeod, the cases occurred in the hot, rainy, and cold seasons, but those in the cold season presented 'severer throat symptoms and a more acute character generally.'[4] As regards age there seems difference of opinion: some observers describe it as most common among adolescents and adults, while Professor Thomas, on the other hand, describes it as 'especially a disease of childhood, attacking indiscriminately boys and girls, older and younger children down to sucklings.' He, however, adds that 'adults up to about forty years are not unfrequently affected.' Sex is said to have little influence on its incidence. It is described as occurring in the British Isles, on the Continent, in Egypt, in India and America. 'Necessary relations between epidemics of rubeola and those of other infectious diseases,' according to Thomas, 'do not exist.'

The consensus of modern authorities as to the existence of a malady of the above sort seems to be too strong to allow of serious doubt upon the point; but to the independent mind a perusal of the literature of the subject may perhaps justify suspicion that the attitude adopted by some authorities upon this matter is somewhat too exclusive and absolute. It has to be noted that some of the epidemics which have been recorded as examples of this disease, by apparently careful observers, have differed conspicuously from the characters now generally regarded as indicative of the malady in question. In some of such epidemics a considerable mortality seems to have occurred, and in other respects the disease has differed from that now regarded as true rötheln. It is easy, of course, to assert that such outbreaks as do not exactly coincide with the modern conception of rötheln are but unrecognised outbreaks of either scarlet fever or measles, but there is a decided suspicion of *petitio principii* about such a procedure. It may, for instance, be that rötheln, admitting it to be a separate disease, is susceptible, under certain conditions of environment, of greater variations than it is customary to admit—indeed, more recent experience seems in favour of this being the case. Or, again, it may be that in addition to the separate malady having the characters usually ascribed to rötheln, there are other maladies also capable of separation from measles and scarlet fever. In a sense, too, the separate and specific character of rötheln is perhaps exaggerated, for it seems now to be usually held that it has no definite re-

[1] William Squire, M.D., article on 'Rubella,' *Quain's Dictionary of Medicine*, vol. ii. Dr. Clement Dukes also found that thirty-nine out of sixty-three cases had previously had measles.

[2] Fagge, *op. cit.*, i. 224.

[3] See Dr. Hopwood's article on Rötheln in Fowler's *Dictionary of Practical Medicine*.

[4] 'On the Prevalence of Epidemic Roseola in Calcutta,' by Kenneth McLeod, A.M., M.D., F.R.C.S., *Epidem. Soc. Trans.*, 1884–85.

lationship to ordinary measles. This of course may be so, but the two diseases would certainly seem to bear a strong family resemblance, and it would appear not unlikely that rötheln, although a relatively separate and moderately stable malady, may still be genetically related, in the sense at least of descent from a common stock, to measles or scarlet fever—probably the former. Such other epidemics, too, as those above referred to, which seem to have differed somewhat from ordinary scarlet fever and measles, as well as perhaps from rötheln, may possibly have been still other descendants from the common stock; or they may perhaps have been even more closely related to measles or scarlet fever, from one or other of which maladies they may have been recently evolved under special circumstances of environment.

Neither would there seem any inherent impossibility in the notion of some of such disease varieties, including rötheln, being of a hybrid character, as has frequently been suggested.

It might be suggested as a criticism upon the hybrid theory that in bacteria sexual processes are absent. This, however, can by no means be safely affirmed.

Neither scarlet fever nor measles appears decidedly to protect against rötheln, nor does that disease, it is said, confer any immunity against them. But it must be borne in mind that protection is a relative matter; and it does not follow, because in one epidemic of rötheln a certain number, and even a comparatively large number, of persons attacked have previously suffered from measles, that the escape of some other persons in such epidemic may not have been due to protection afforded by previously acquired measles. If it is true that 'more escape in a house or school during an epidemic of rubeola than during one of measles,' this may possibly, though of course not necessarily, be due to the cause referred to. Lastly, it would be premature to allege that diseases descended from a common stock must necessarily protect against each other.

Whatever its origin, however, there seems no doubt, as already said, that cases, both sporadic and epidemic, of an ailment, having some of the appearances of measles and some of those of scarlet fever, are frequently met with in this and other countries; but at present little can be said with confidence as to their epidemiological characters, and the whole subject requires careful study.

Even Professor Thomas, one of the most distinguished and ardent supporters of the separate and specific character of this disease, remarks: 'There are few diseases in regard to which opinions vary so much as about rubeola, or rather about that which is designated as rubeola by various authors. The work of separation and discrimination, which has by degrees given us a definite conception of the other acute exanthems, has by no means reached its final result as regards this.'

There is one matter, however, which, from a public health point of view, is important, and that is the certainty that in practice 'rötheln' is too frequently pressed into the service as a cloak to difficulty in diagnosis. '"German measles,"' says Dr. Goodhart, 'is a term which is terribly abused. A doubtful rash makes its appearance, and the medical man, instead of saying he is not certain of its nature, calls it German measles. "Then it is not scarlatina?" ask the parents. "No," says the doctor; and the parents, thinking nothing of measles, take no precautions. Any hospital physician sees many such cases, and knows also very well—considering the rarity of the actual disease—that, when he has to do with what is called German measles, it is more probable than not that the nature of the malady is scarla-

tinal, and that in this direction he must look for the explanation of whatever sequelæ he may meet with.' The disastrous consequences which follow the course above indicated need not be pointed out.

SCARLET FEVER

Synon.: *Scarlatina*; Fr. *Scarlatine*; Ger. *Scharlachfieber*; It. *Febbre Scarlatina.*

History and Geographical Distribution.—The complete differentiation of scarlet fever was set on foot by Sydenham, whose description of the disease as 'febris scarlatina' was based upon experiences of it as it occurred in London from 1661 to 1675. In this connection it is interesting to note that Sydenham makes no mention of sore throat as one of the symptoms of scarlet fever (Fagge). Subsequently the distinct and separate character of scarlet fever was more fully established by Heberden, Fothergill, Willan, and Withering. But though not differentiated until the latter half of the seventeenth century, scarlet fever was described by Ingrassias as early as 1550, and somewhat later by Döring, Sennert, and other writers. According to Hirsch,[1] 'the oldest notice relating probably to an epidemic of scarlatina dates from Sicily, 1543;' but Hirsch is of opinion that the disease was prevalent on the continent of Europe long before the period from which we derive the earliest medical accounts of it.

Prior to the time of Sydenham, scarlet fever had been regarded as a variety of measles, and even Morton, a contemporary of Sydenham, held to this view and described it as '*morbilli confluentes.*'

Though it is evident from what has been said that scarlet fever has long been prevalent in Europe, its introduction into many other parts of the world seems, as in the case of measles, to have taken place in comparatively recent times. Thus it was apparently unknown in North America until about 1735, and it did not become prevalent in South America until 1830. It first reached Australia and Polynesia in 1848 (Hirsch).

At the present day scarlet fever is most widely distributed in the north-western countries of Europe. In Russia also it appears to be somewhat widely prevalent. It occurs, though apparently to a lesser extent, in Italy, Turkey, Greece, and some of the Mediterranean islands. It is widely diffused over North and parts of South America, but it does not appear to have been frequently prevalent in Australia; and, although often imported, it has never firmly established itself in Asia, except along the coast of Asia Minor, or in Africa except perhaps in Algiers. As regards the broad geographical distribution of scarlet fever, Hirsch remarks that 'the area of diffusion of scarlet fever is much smaller than that of small-pox or of measles; that the continents of Asia and Africa, which, as we have seen, are among the chief seats of these two diseases and especially of small-pox, have been visited by scarlet fever at the utmost to a very small extent, allowing even for imperfections of record.'

As regards England, the Registrar-General points out in his 47th Annual Report (page xiv) that for the years 1871–80 the mortality from scarlet fever among children under five years of age was above the general average in the following counties, beginning with those having the highest mortality: Durham, Lancashire, Northumberland, West Riding, Staffordshire, War-

[1] See Hirsch, *op. cit.*, i. 172.

wickshire, Cheshire, South Wales, Worcestershire, Derbyshire, and Cumberland. 'It thus appears,' he remarks, 'that even after due correction is made for age distribution, scarlet fever is for some reason or other most destructive in the industrial, and especially the mining counties. . . . The explanation that naturally suggests itself is that probably the population in industrial and mining counties live in more than averagely close aggregation, and that the spread of infection is thus facilitated.'

'If, however, this were the true and complete explanation we should expect the geographical distribution of other infectious diseases to tally with that of scarlet fever.' But this, the Registrar-General remarks, 'is not true as regards diphtheria; nor does it seem altogether true as regards measles.'

The contrast between the geographical distribution of diphtheria and scarlet fever is a matter of very great interest, and has also been especially pointed out by Dr. G. B. Longstaff,[1] and by Dr. Edgar Barnes,[2] who has published maps showing the marked difference between the distribution of diphtheria and scarlet fever during the years 1880–86.

Periodicity.—Although more or less constantly present in large communities in which it is firmly established, scarlet fever at times assumes a wide epidemicity. It extended, for instance, over Denmark, England, Germany, and France in 1825–26, and again, as well as over Ireland and Russia, in 1832–35; over Germany, Denmark, England, and Scotland in 1846–49; over North America in 1821 and 1851, and South America in 1831–37 (Hirsch). From a study of the Swedish mortality records Dr. Arthur Ransome considers that, as regards scarlet fever mortality, 'not only a short cycle of four to six years may be traced, but also a long undulation of fifteen or twenty years or more; which may be likened to a vast wave of disease upon which the lesser epidemics show like ripples upon the surface of an ocean swell.'[3]

Dr. Whitelegge finds that at Nottingham scarlet fever exhibits a weekly cycle, the notified cases falling to a minimum upon the Wednesday. This he regards as probably due to the diminished likelihood of infection through the agency of school attendance upon the Sunday.

Mortality.—The following table, compiled from the annual reports of the Registrar-General, shows year by year the mortality from scarlet fever in England and Wales, during registration times.

It will also be seen that diphtheria deaths were included by the Registrar-General with those from scarlet fever until the year 1855. But subsequently to that date the returns form a continuous series comparable with one another *quâ* scarlet fever; and it will be at once observed that there has of late years been a most satisfactory decline in the scarlet fever death-rate. It must not, however, be too confidently assumed that this decline is permanent, for, as Dr. Thorne[4] remarks: 'There is, perhaps, no disease concerning which it can be said with less certainty that diminutions year by year in its fatality foretell a permanent lessening in its prevalence than is the

[1] The Geographical Distribution of Diphtheria in England and Wales. Sup. Report of the Medical Officer to the Local Gov. Board for 1887. Republished in 'Studies and Statistics.'

[2] 'The Etiology of Diphtheria,' by E. G. Barnes, M.D., *British Medical Journal*, July 28, 1888.

[3] Arthur Ransome, M.D., 'On the Form of the Epidemic Wave, &c.,' *Trans. Epidem. Soc.*, 1881–82.

[4] R. Thorne Thorne, M.B., F.R.S., 'The Progress of Preventive Medicine during the Victorian Era.' Being the Inaugural Address delivered before the Epidemiological Society of London, Session 1887–88.

TABLE II.—*Showing year by year the total deaths registered in England and Wales during registration times as due to scarlet fever; with the corresponding death-rates per million living and the average annual death-rate for each quinquennial period.*

England and Wales				England and Wales			
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1838	5,802	380		1865	17,700	837	982
1839	10,325	666		1866	11,685	546	
1840	19,816	1,256	797	1867	12,300	567	
1841	14,161	889		1868	21,912	996	960
1842	12,807	794		1869	27,641	1,244	
1843				1870	32,543	1,446	
1844				1871	18,567	815	
1845				1872	11,922	515	
1846				1873	13,144	562	759
1847	14,697	857		1874	24,922	1,050	
1848	20,501	1,178	884	1875	20,469	851	
1849	13,123	747		1876	16,893	691	
1850	13,370	753		1877	14,456	585	
1851	13,634	758		1878	18,842	753	680
1852	18,887	1,035		1879	17,613	694	
1853	15,699	853	907	1880	17,404	675	
1854	18,528	995		1881	14,275	548	
1855	16,929	894		1882	13,732	521	
1856	13,557	705		1883	12,649	475	436
1857	12,646	652		1884	10,863	402	
1858	23,711	1,212	806	1885	6,355	233	
1859	19,310	976		1886	5,986	218	
1860	9,681	485		1887	7,859	282	
1861	9,077	451		1888	6,378	226	241
1862	14,834	728	982	1889	6,698	235	
1863	30,475	1,478		1890	6,974	242	
1864	29,700	1,418		1891	4,959	171	

Including Diphtheria

case in regard of scarlatina. Not only do different outbreaks vary very greatly as regards mortality, but the epidemic prevalences tend to occur in cycles; and an abatement extending over a few years has been known to be followed by a wide and fatal diffusion of the infection. And not only so, but the more recent diminution in the amount of fatal scarlatina may be in noteworthy part matter of diagnosis. . . . But, after making every allowance, there remains the important fact, that ever since the decennial period 1861–70 there has been a very general and fairly steady diminution in the fatality of scarlatina in this country, until, in 1885, the rate of death from that cause was less than a quarter of that which formerly prevailed; and it is impossible not to regard so long-continued and marked an abatement as an indication that some of the means conducing to the spread of this very fatal fever have been materially restricted.'

Fatality or Case Mortality.—The fatality of scarlet fever varies largely in different epidemics and even at times during the different stages of the same epidemic. It may range between 3 and 30 per cent. of attacks. Probably 10 per cent. is near the average rate for hospital practice; but since hospital cases are doubtless above the average as regards severity, especially if allowance is made for the mild cases among the general public which escape identification, it is likely that 10 per cent. is too high as a general estimate.

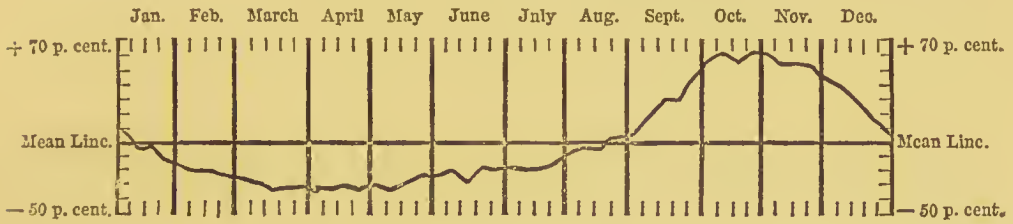
Of the causes of the decided variations in the malignancy of different epidemics little is known. Judging from the evidence, it would seem clear

that although, other things being equal, unwholesome conditions of life almost certainly have some influence in this respect, and the same is probably true of season of the year, yet these are far from being the controlling factors.[1] The type of an epidemic is, in fact, evidently something much more fundamental—something, we may hazard the suspicion, intimately concerned with the evolutionary phases of the species of micro-organism upon which the disease depends.

Influence of Climate.—It appears highly probable that scarlet fever is influenced by climate. Not only is it clear, as will be shown later, that in this country its prevalence is to a considerable extent regulated by season, which is suggestive of the influence of varying climatic conditions, but the fact that the disease has not established itself in the tropical or sub-tropical portions of Asia and Africa would certainly seem to imply that there is something in the climate of those countries which is unfavourable to its development. It would not by any means appear to exclude this inference to refer, as Hirsch does, to the fact that scarlet fever 'has often been found epidemic in the tropical countries of South America,' for climate includes many factors other than mere temperature. The subject requires much closer study before a definite conclusion can be reached; but it is probable that temperate and humid climates are most favourable to scarlet fever.

And Season.—As regards season, Hirsch found that of 435 epidemics of scarlet fever in Europe and North America, 29·5 per cent. attained their maximum in the autumn, 24·7 in winter, 24 in summer, and 21·8 per cent.

Scarlet Fever, for all Ages and both Sexes.



in spring. In London, according to Dr. Buchan and Sir Arthur Mitchell, whose curve is here given, the mortality from scarlet fever is at its lowest in April, and attains its maximum in October, again falling rapidly in December.

It must not, however, be assumed that the seasonal prevalence of scarlet fever is the same for all countries. According to Dr. Whitelegge, for instance, the mortality curve for New York is practically the reverse of that for London, the maximum being in April, and the minimum in September.

But for England it may be confidently stated that both the prevalence and mortality of scarlet fever are greatest in the autumn and least in the spring.

Dr. Ballard found that of 3,850 cases of scarlet fever recorded in the books of the Poor-law medical officers and the various medical institutions in Islington for the twelve years 1857–68, 17·7 per cent. occurred in the first quarter, 17·3 in the second, 29·9 in the third, and 35·1 in the fourth.

Dr. Longstaff[2] has constructed the following diagram, which shows the close similarity between the curve based upon the weekly average of London scarlet fever deaths and that of the admissions to the Metropolitan Asylums Board hospitals of scarlet fever patients, for the ten years 1875–85.

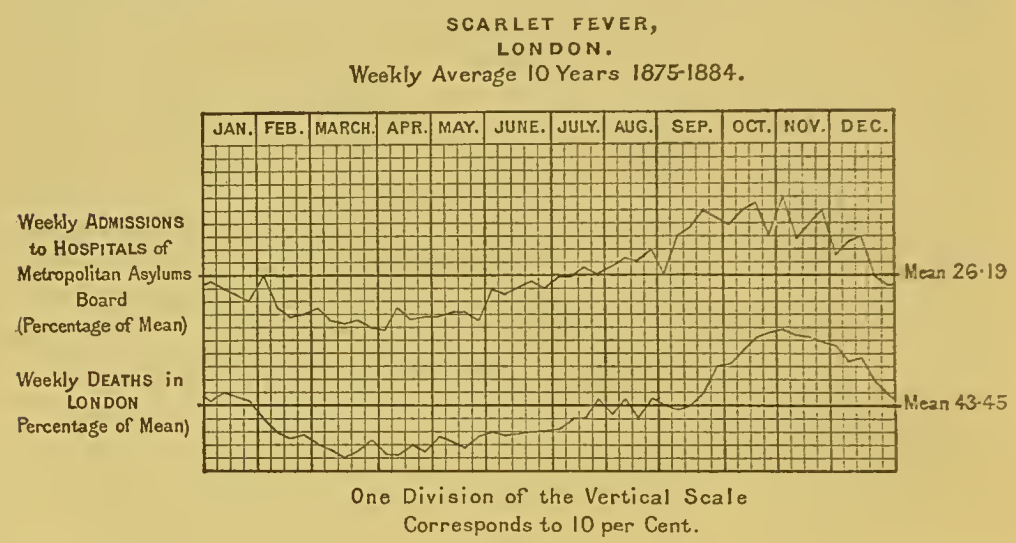
It must be remembered, however, that the admissions to hospital must not offhand be assumed to be a correct measure of the general scar-

[1] See Hirsch, *op. cit.*, i. 187.

[2] *Studies in Statistics*, p. 405. Reproduced by Dr. Longstaff's permission.

latinal prevalence, for it is doubtless the more severe cases that seek admission to hospital. But this possible source of fallacy is in great part eliminated by a table constructed by Dr. Whitelegge, which is based on the notification returns of twelve large English and Scotch towns, and which also shows the prevalence of scarlet fever to be greatest in the autumn.[1]

As to the influence of particular meteorological conditions upon scarlet fever prevalence and mortality, the evidence put forward by different observers is conflicting. But Dr. Longstaff [2] has pointed out that there appears to be an inverse relationship between the mortality from scarlet fever, and certain other diseases, and the rainfall. 'The same result is obtained,' he says, 'whether the amount of the fall or the number of days on which it falls



is considered, the connection being somewhat closer in the latter case.' This accords with Dr. Ballard's general conclusion, that a temperature above the average for the season, and a dry state of the atmosphere with little rain, favour the prevalence of scarlet fever more than the reverse conditions.

From what has been said above, it is clear that in this country both the prevalence and mortality of scarlet fever are greatest in the autumn and least in the spring. But what shall we say as to fatality, i.e. the proportion of deaths to attacks? Is scarlet fever more fatal as well as more prevalent in autumn than in other seasons? Distributing into the seasons in which they happened the 1,993 scarlet fever deaths which occurred in the entire parish of Islington during the twelve years 1857–68, and distributing also in similar fashion the 3,850 cases of scarlet fever which occurred in the public practice of the district during the same period, Dr. Ballard, by calculating for each quarter the proportion borne by the deaths in the entire parish to the cases in the whole public practice, arrived at the conclusion that in Islington parish, during the twelve years in question, scarlet fever was, on the whole, less fatal and serious during the third or summer quarter, and most fatal in the first or winter quarter, and that the fatality of the spring and autumn was equal.

Dr. Whitelegge, again, points out that the seasonal curve of notified attacks differs but little in outline from the mortality curve, but that at almost every point the deviation from the mean line is less in the death curve than in the attack curve; in other words, the mortality rises and falls proportionately less than the cases do, indicating that at the season of the year in which the disease is most prevalent it is least fatal, and *vice versâ*.

[1] *Epidem. Soc. Trans.*, N.S., vol. vii. [2] *Op. cit.*, p. 322.

On the other hand, some observers have been led by their personal experiences to regard the autumn as the season in which scarlet fever is most severe as well as most prevalent. Thus Dr. Gresswell [1] quotes Fothergill, to the effect that he met with more cases of malignant scarlet fever 'from September to December inclusive than in all the other months together.' Willan wrote of scarlet fever: 'This disease, as is usual in the summer months, exhibits a mild train of symptoms, and soon terminates favourably, without producing any material affection of the throat. For some years past it has always been most virulent and dangerous in the months of October and November, but generally ceased on the first appearance of the frost.' Other observers have found 'that in summer the affection of the throat was frequently so trifling as not to demand any particular attention. Haygarth found the disease most virulent in October and November.'

As regards the 588 patients (admitted to the Asylums Board hospitals between early September 1887 and early February 1888), of whom Dr. Gresswell had sole charge, it appears that the severity of the disease 'was greatest among persons attacked in October, and the first three and a half weeks or so of November; and that it began to fall off rapidly afterwards.' This increased severity made itself apparent not only in the death returns, but in the relative frequency of the graver manifestations of the disease. 'I shall show,' says Dr. Gresswell, 'that after a certain date the symptoms, which had up to that date evidenced a general progressive increase of relative frequency and intensity, remarkably ameliorated; and that this amelioration commenced definitely in the latter part of November.' Dr. Gresswell then illustrates by statistical table, and graphically by a chart, the relation to season of the complications and more serious phases of the disease as observed in his 588 patients. In reference to the table and chart he points out that 'though in some points the clinical statistics of the September, October, and November cases were much alike, the relative incidence of many complicating conditions increased from September into October or November, and fell away suddenly in December. . . . The graver manifestations, in fact, became relatively more frequent, and I may add more marked, from September into October or November; and then they suddenly fell off; not merely in regard to two or three determinations, but in regard to most of them—changes which were also expressed in the monthly mortality.'

It has to be borne in mind, of course, that Dr. Gresswell's scrutiny of this question only extended to a limited number of cases at a particular period of an epidemic; and whether further inquiry, of similar exactitude, based upon a larger series of attacks, will support the view that the fatality of scarlet fever, like its prevalence and mortality, is greatest in the autumn months remains to be seen.[2]

But that the *prevalence and mortality* of scarlet fever in this country are at their lowest in the spring, and thenceforward rise steadily through the summer, attaining their maximum in the autumn, we have seen to be facts beyond doubt.

The Influence of Age and Sex.—The relations of age and sex to attack and death by scarlet fever are dealt with at length in the 49th Annual Report

[1] *Natural History of Scarlet Fever.* By D. Astley Gresswell, M.A., M.D.

[2] Since the above was written, Dr. Whitelegge has explained the results of his further researches into the subject of epidemic disease periodicities. These results tend to confirm his previous suspicion that the seasonal increase in the mortality of certain diseases, including scarlet fever, is due to increased prevalence of such diseases, and not to an increase in their fatality, i.e. case mortality.

(1886, page xiv) of the Registrar-General. It is impossible here to give the details of the Registrar-General's investigation, but the important conclusions at which he arrives are thus stated :—

'1. The mortality from this disease is at its maximum in the third year of life, and after this diminishes with age, at first slowly, afterwards rapidly. 2. This diminution is due to three contributory causes : (*a*) the increased proportion in the population at each successive age period of persons protected by a previous attack ; (*β*) the diminution of liability to infection in successive age periods of those who are as yet unprotected ; (*γ*) the diminishing risk in successive age periods of an attack, should it occur, proving fatal. 3. The liability of the unprotected to infection is small in the first year of life, increases to a maximum in the fifth year or soon after, and then becomes rapidly smaller and smaller with advance of years. 4. The chance that an attack will terminate fatally is highest in infancy, and diminishes rapidly with years to the end of the twenty-fifth year, after which an attack is again somewhat more dangerous. 5. The female sex throughout life, the first year possibly excepted, is more liable to scarlet fever than is the male sex. 6. But the attacks in males, though fewer, are more likely to terminate fatally.

'Now it is sometimes said that the separation from its family of a child who is attacked by scarlet fever is scarcely worth the trouble and expense it involves, seeing that the rest of the children, though they may escape on that special occasion, are almost certain to contract this very common disease at some future time, and may therefore as well, if not preferably, have it at once. The results, however, to which our statistical inquiry has led us, are completely subversive of such a position. They show—independently of the plain fact that a very large proportion of persons go through life without ever contracting the disease—that the longer an attack is deferred, the less likely it is to occur at all ; and not only so, but that, even supposing it to occur eventually, the less likely it is to end fatally.'

The influences of age and sex in relation to scarlet fever have also been dealt with by Dr. Whitelegge[1] upon the basis of 6,288 cases of scarlet fever notified in the three large towns of Nottingham, Salford, and Leicester. The results, thus independently arrived at, substantially coincide with those of the Registrar-General, except that in Dr. Whitelegge's cases the case mortality was rather higher in the second year of life than in the first. The practical conclusion from Dr. Whitelegge's investigation is that 'in shielding a child against infection during the first few years of life there is a double gain ; every year of escape from scarlet fever renders him less and less susceptible, until finally he becomes almost insusceptible ; and, secondly, even if he should ultimately take the disease, every year that the attack is deferred reduces the danger to life which it brings.'

Cause and Dissemination.—That the essential cause of scarlet fever is a micro-organism there can, on general principles, be little doubt ; but with respect to this branch of the subject, see p. 152, *ante*. As regards dissemination, it has long been known that scarlet fever may be spread by direct infection from case to case, and also by indirect infection through the medium of infected clothing, furniture, books, letters, &c. There are cases on record, too, which seem to place beyond doubt the possibility of the poison being handed on from infected linen to other linen in laundries, and in this doubly indirect way spreading the disease. Moreover, it has frequently been observed that the scarlet fever poison is capable, under certain circumstances, of retaining its infective power for considerable periods of time, altogether

[1] *Epidem. Soc. Trans.*, N.S., vol. vii., 1887–88.

outside the human body. A fresh outbreak of the disease, for instance, has been observed to follow upon the disturbing of some previously infected garment which had been put away in a drawer for months.

Until recent years the above methods of dissemination, i.e. immediate infection from a previous human case, or mediate infection from antecedent human cases, through the agency of fomites, were generally regarded as exhausting the possible methods of scarlet fever transmission. And farther it was apparently not contemplated that the mediate method referred to involved any multiplication of the organism outside the human body, but simply its storage and conveyance. In the year 1870, however, Dr. M. W. Taylor opened the way to a very great advance in our knowledge of the natural history of scarlet fever. While studying an outbreak of that disease which occurred in the town of Penrith, he observed that the main incidence of the disease was upon the customers of a particular dairy. Following up the suggestion thus gained, he found that, prior to the general outbreak in the town, a child at the dairy in question had suffered from scarlet fever, and that not only was the milk taken into the infected dwelling before distribution to the customers, but that the child's mother, while acting as sick-nurse, also at times milked the cows. These facts, coupled with the circumstance that many of the sufferers in the town had no communication with the dairy except through the medium of the milk, seemed irresistibly to point to milk as the vehicle of the infection. Subsequently other observers recorded like experiences elsewhere, and as a result Mr. Ernest Hart was able, in 1881, to refer to fifteen epidemics of scarlet fever in which there was evidence of dependence upon infected milk service. So far, however, the milk was generally looked upon as having become accidentally infected by human agency. Later investigations by Mr. Power and Dr. Klein seem to have shown beyond doubt that human scarlet fever may be produced by milk which owes its infective property to an ailment of the cow. But with respect to this, and the subject of milk-scarlet fever generally, the reader must be referred to Dr. Klein's article (p. 146, *ante*).

The infection of scarlet fever seems to be given off by the breath, the secretions from the nose, mouth, pharynx, ears, and perhaps kidneys, and also by desquamating cuticle. It may apparently cause disease either by being inhaled or swallowed. There is no evidence of its being conveyed by water, and inasmuch as the disease does not appear to spread in the neighbourhood of fever hospitals, it would seem that the infection is not capable of being conveyed any great distance by air currents.

According to Prof. Thomas, several observers have successfully inoculated persons with scarlet fever virus, the inoculation being in some cases followed by general, and in others by local scarlet fever. Children so inoculated were said not to contract the disease when subsequently exposed to infection. He also states that a disease corresponding to scarlet fever in man has been met with by different observers in the horse, 'cats, dogs, swine, and other domestic animals.' Attacks of what appears to be scarlet fever occasionally follow surgical operations, and it is usually said that such injuries increase susceptibility to that disease. At the same time it must be remembered that some of such cases are probably not scarlet fever at all, but septic conditions. Cases of the kind have been observed under circumstances which seemed to put scarlet fever infection out of the question. It appears, too, according to McLeod,[1] that so-called surgical scarlet fever is met with in India, where scarlet fever is practically unknown.

Period of Incubation and Infectiveness.—The period of incubation of

[1] *Epidem. Soc. Trans.*, N. S., vol. iv., 1884-85.

scarlet fever is almost always, if not invariably, less than a week, and it may be as short as a few hours. Usually, however, it is somewhere between two and four days.

The infective period extends from the earliest symptoms to the end of convalescence, but it is greatest when the fever is at its height. No patient can ever with safety be allowed to mix with the healthy until the expiration of at least six weeks, no matter how slight the attack; and it will often be necessary to extend the period of isolation to eight or nine weeks, or even longer, for it must be continued until all traces of desquamation have ceased. Moreover, it must be remembered that discharges from the ear and nose are capable, for a time at least, of spreading the disease, and the same is possibly true of albuminuria. It also has to be borne in mind that recrudescence of the disease—especially recurrent sore throat and albuminuria—sometimes occurs, and it is doubtful how late such true recrudescences may take place, and renew the infective condition of the patient.

Protection.—One attack usually confers immunity throughout life, though well-marked second, and even third, attacks occasionally occur. According to Dr. Squire,[1] persons too who have previously suffered from the disease, 'when much exposed to it, may have sore throat or other signs of partial sickening sufficient to start the disease elsewhere.' Such modified second attacks quite likely renew the protection of the persons thus affected.

Relation to other Diseases.—It has been stated by Löschner, Köstlin, and others that scarlet fever epidemics especially tend to follow upon outbreaks of measles. This, however, is denied by other authorities. If any relationship of the kind, beyond a purely casual one, occurs, the probable explanation would seem to be that suggested by Köstlin, viz. that measles increases the susceptibility to scarlet fever, and, indeed, this would appear not to be unlikely.

Scarlet fever is sometimes found closely associated with diphtheria.

The belief in a close relationship between scarlet fever and a form of puerperal fever, in the sense of the infection of the former being capable of originating the latter, has been widely held. It is certain that in some cases in which recently confined women have been freely exposed to the poison of scarlet fever, no harm has resulted. Nevertheless, the danger of such an occurrence has been maintained by high authorities, and is probably a real one, even if it may have been somewhat exaggerated. It derives some indirect support, too, from the circumstance that scarlet fever seems undoubtedly to exercise an injurious influence upon vaccination, apparently giving rise to irregularity, and in some cases playing a part in the causation of septic mischief with erysipelatous manifestation.

TYPHUS FEVER

Synon.: *Petechial Fever, Gaol Fever, Ship Fever, Spotted Fever.* Fr. *Typhus*; Ger. *Exanthematischer Typhus, Flecktyphus*; It. *Tifo.*

History and Distribution.—Typhus has only in our own day been clearly differentiated from other fevers, notably enteric and relapsing fevers, with which it had been previously confused. There can be no doubt, however, as to its having existed for a number of centuries, and Murchison believed it to have been referred to by Thucydides. Hirsch, after pointing out that much of

[1] 'Scarlet Fever,' *Quain's Dict. of Medicine.*

the 'war or famine sickness' of history was doubtless 'a mixture of various kinds of disease, such as diarrhœa, dysentery, scurvy, typhus, and frequently also malarial fever and typhoid,' refers to an outbreak of fever at a monastery near Salerno in the year 1083 as one of the earliest records of typhus having any degree of definiteness. The more reliable history of typhus, however, seems to date from the sixteenth century, during which period Hirsch finds 'numerous accounts of the disease from almost every part of Europe.' Throughout the seventeenth, eighteenth, and earlier part of the present century it appears to have been widely prevalent in Europe, visiting at one or another time practically every country, including Iceland. Great Britain and Ireland suffered extensively; and indeed Ireland occupies a conspicuous place in the history of this disease. 'In no part of Europe,' says Hirsch, 'does typhus bear the character of an endemic malady so decidedly as in Ireland.' Severe epidemics, many of which appear to have spread from that country to England and Scotland, occurred in Ireland in the years 1708–10, 1718–21, 1728–31, 1770–72, 1797–1802, and during the present century 1816–19, 1821–22, 1826–28, 1836–37, 1846–47, 1862–64. Russia, Italy, and Spain have also suffered heavily. Hirsch considers that, speaking generally, the 'period of typhus' came to an end about the year 1815, and he states that since that time 'the disease on European soil has only once, in 1846–47, attained the same general diffusion which the history of pestilence presents to us so often in former centuries.' During more recent years the prevalence of typhus fever in Europe has markedly abated, and the disease has tended more and more to limit itself to particular areas.

Beyond Europe, typhus has prevailed more or less extensively in Persia, North China, Egypt, and North America, in which latter country it would seem to have been largely a result of immigration. On the other hand, Japan, Australia, New Zealand, and the greater part of Africa, including Cape Colony, are said to have been exempt from the disease. In India the disease seems to be at times observed.

The most important fact brought out by a study of the history of typhus fever is its practically invariable association with overcrowding, starvation, and general misery, whether as a result of war, famine, or more continuous social defects in the communities affected. This is seen to be the case both by a wide survey of the history of the disease and by a study of its behaviour in a given country. But this matter will be referred to later.

Mortality.—The more recent history of typhus fever in England may be sufficiently inferred from Table III. (p. 276), in which it will be seen that with the exception of a temporary check in 1882–83 the registered mortality from this disease has steadily declined.[1] The figures in the table are taken from the Annual Report of the Registrar-General for 1890 (pp. xxxviii–xl). The typhus deaths were not abstracted prior to 1869.

Case Mortality.—As will be seen immediately, age is such an important controlling factor as regards the fatality of typhus fever, that the two matters cannot be usefully discussed apart. Murchison, however, found that of 18,268 cases of typhus at all ages admitted into the London Fever Hospital, 3,457, or 18·9 per cent., ended fatally. But he points out that these, being hospital cases, were doubtless above the average as regards severity, and he

[1] It is doubtful, however, whether the actual typhus mortality during recent years is not somewhat understated in these death returns. An inquiry in the winter of 1886–87 by Mr. Spear showed typhus to be existing in no less than seventeen English towns, and in many instances the deaths had been returned as due to other causes. See later. But notwithstanding this there is no doubt that a marked abatement has occurred in recent years.

gives 10 per cent. as a general estimate of typhus fatality. But this will vary in different epidemics.

TABLE III.—*Showing the total Deaths from Typhus Fever for each Year from* 1869 to 1891 *and the corresponding Death-rate per Million living.*

England			England		
Year	Total deaths	Death-rate per million living	Year	Total deaths	Death-rate per million living
1869	4,281	193	1881	552	21
1870	3,297	147	1882	940	36
1871	2,754	121	1883	877	33
1872	1,864	80	1884	328	12
1873	1,638	70	1885	318	12
1874	1,762	74	1886	245	9
1875	1,499	62	1887	211	8
1876	1,165	48	1888	160	6
1877	1,104	45	1889	137	5
1878	906	36	1890	151	5
1879	533	21	1891	137	5
1880	530	21			

Influence of Race, Age, and Sex.—There is no sufficient evidence that race exercises any influence over liability to typhus. It is true that the Irish have suffered excessively from this disease, but the circumstance is probably due to the relatively unwholesome conditions under which the poor of Ireland habitually seem to have lived, whether at home or abroad.

No age is exempt from typhus fever, but persons between the ages of fifteen and twenty years are most liable to attack. As regards fatality, however, the matter is very different, and the risks to life, in the event of attack, increase largely with age.

Murchison gives the case mortality of the higher ages as 35·39 in persons between 30 and 40; 43·48 in persons between 40 and 50; 53·87 in those between 50 and 60; and 67·04 in those over 60. As regards the earlier periods of life he says: 'The rate of mortality [fatality] was somewhat greater during the first than during the second ten years of life. Thus, the mortality during the first five years of life was 6·69 per cent.; in the second lustrum it fell to 3·59; between ten and fifteen it was only 2·28 per cent.; and between fifteen and twenty 4·46 per cent. After twenty it went on progressively increasing.'[1]

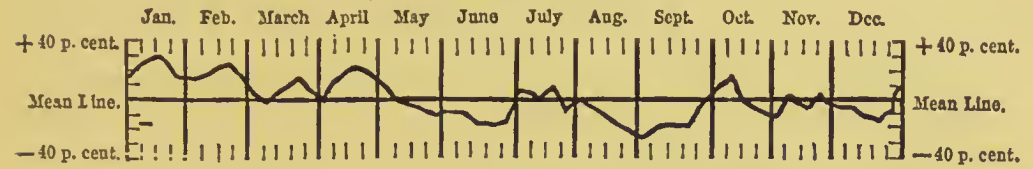
Sex is said to have little influence upon liability to attack, and the total deaths are also pretty equally divided between the two sexes. It would appear, however, that the fatality is usually somewhat greater for males at all ages taken together, than for females. Murchison's cases gave a male fatality of 19·6 per cent. of attacks, and a female fatality of 18·2 per cent. From five to fifteen years the fatality among females was twice as great as that among males, but after fifteen years the male fatality was the greater at every period of life.

Influence of Climate and Season.—From what has already been said with regard to the history and distribution of this disease, it will have been seen that it is mainly a disease of temperate and colder climates; for, although it is true that it has prevailed in some tropical countries, it seems even there to have occurred in the colder seasons of the year, and in the more elevated localities. In England both the prevalence and mortality of

[1] *The Continued Fevers of Great Britain*, p. 236. By Charles Murchison, M.D., F.R.S. 3rd ed.

the disease have probably, upon the whole, been greater in the late autumn, winter, and spring, than in the summer and early autumn.

Typhus—for all ages and both sexes (*Buchan and Mitchell*)
(London deaths, 1869–1874)



As regards prevalence, this is attested to by the records of admissions to fever hospitals. As regards mortality, the same result is indicated in the above curve.

Dr. Longstaff[1] found that for the ten years 1875–84 the mortality was above the mean from January to April inclusive, again slightly in June, and lastly through October, November, and December. And the maximum was in November instead of January as shown above. On the whole, therefore, it would seem that typhus bears a less constant relation to season than is the case with several of the other epidemic diseases.

Cause and Dissemination.—It is inferred on general grounds that typhus fever is caused by a micro-organism, but so far this has not been established.

With regard to accessory causes, we have already seen that history discloses an association between destitution and overcrowding, and the rise of outbreaks of typhus, so close as to render it impossible to escape the view of a causal connection subsisting between the two phenomena. Indeed, so striking is the association that it has frequently been maintained, and among others by no less an authority than Murchison, that the disease may arise *de novo* from such conditions alone.

In the introductory section of this paper an attempt has been made to show that such a doctrine, if intended to signify the origination of the assumed microphytic cause of disease independently of parent microphytes, is untenable. But several other modes, by which a disease, as such, might practically have *de novo* origin, have also there been indicated, and it is possible that one or other of such ways may have to do with the beginning of typhus.

Another matter of interest with respect to the origin of typhus outbreaks is the possibility of their being initiated by a 'revivification of long dormant contagion,' and Mr. Spear states that he found reason for attributing recent disease to this cause.

But whatever may be the truth as to the life history of the typhus virus, there is no question of the influence of overcrowding and poverty as most important etiological factors in this disease. All authorities are agreed upon that point, and it may be confidently asserted that, apart from such circumstances, typhus would never arise. When once started, however, the disease is highly infectious, and although in the main it still tends to cling to the overcrowded homes of the destitute, it frequently passes beyond those limits, for any who are brought within the range of infection may become attacked, and act as foci for the dissemination of the disease elsewhere. Fortunately there is a circumstance with respect to this infection which largely tends to limit the ravages of the disease, and that is that it does not appear capable of travelling far through the air without losing its potency. Although highly active in the immediate vicinity of the patient, it soon appears to be rendered harmless by diffusion through the atmosphere. With abundant ventilation

[1] *Studies in Statistics*, p. 402.

of the sick-room, assuming, of course, proper isolation of the sick, there need, therefore, be little danger of the disease spreading, except among the medical attendants and nurses, whose duties necessitate frequent and close contact with the sufferers. That both these classes of persons inevitably incur considerable risk is, however, but too plainly testified to by the records of fever hospitals.

The poison of typhus may be conveyed in clothing, but there is no evidence of its being disseminated by water, milk, food, or domestic animals. It is believed to be given off by the breath and skin of the patient, and typhus patients emit a peculiar odour, perceptible at a distance of a foot or two, which has been thought to be associated with the infection.

Periods of Incubation and Infectiveness.—The period of incubation of typhus fever varies considerably in different cases, and this is probably due to the quality of the poison as regards concentration; probably also to the dose of the poison received and the condition of the recipient. Most authorities, however, including Murchison, give the usual period as about twelve days. Some have stated it as shorter: thus Dr. Beveridge gives it as 'about a week,' and Lebert as five to seven days. Murchison considered that the infection is comparatively slight during the first week of the illness, but that the disease is most contagious from the end of the first week up to convalescence. It is therefore impossible to lay down a definite rule as to the actual length of time that infection will last in different cases, but probably it is never safe to allow a patient to mix with others in less time than a month from the date of attack, and a longer period of isolation will often be necessary.

Protection.—One attack of typhus fever usually protects against the disease for the future; but although second attacks are unfrequent they do occasionally occur, and in exceptional instances the disease appears to confer practically no immunity at all, the individual contracting it whenever exposed to infection. Such cases, however, are very rare.

RELAPSING FEVER

Synon.: *Famine Fever; Seven-day Fever; Bilious Remittent Fever; Bilious Typhoid Fever; Febris vel Typhus recurrens.* Fr. *Fièvre à rechute, Typhus à rechute*; Ger. *Das recurrirende Fieber, Hungerpest*; It. *Tifo Recidivo.*

History and Geographical Distribution.—It is generally considered that the first reliable reference to relapsing fever is that of Rutty, who, in his 'Chronological History of the Diseases of Dublin' (1770), described an epidemic, evidently of this disease, as having occurred there in the year 1739. There can, however, be little doubt that relapsing fever had occurred in earlier times. Murchison, indeed, considered that an epidemic in the island of Thasus, recorded by Hippocrates, 'resembled it very closely in most of its characters, including an intermission of five or seven days between the febrile attacks, jaundice, epistaxis, tendency to miscarry, &c.'

Subsequently to 1739, further epidemics occurred in Dublin, in 1745, 1748, and 1764–65. The first recognisable description of relapsing fever in the records of Scotland seems to date from 1741. In the year 1799, and again in the years 1817 and 1826, the disease occurred in both Ireland and Scotland. It then apparently disappeared until 1841, when it broke out in Scotland, appearing the following year in Ireland (Hirsch). In 1843 it was also widely prevalent in Scotland. So complete seems to have been its cessation in that country between 1826 and this period, tha Murchison says

it was now 'regarded by many as a new disease.' It was at this time that the doctrine of relapsing fever as a distinct and separate disease was definitely promulgated by Dr. Henderson, of Edinburgh, and other Scotch physicians; though, according to Murchison, Dr. O'Brien, of Dublin, had drawn a distinction between relapsing fever and typhus as early as 1828. Prior to this, relapsing fever had most commonly been regarded as a variety of typhus, with which disease it was usually found to be associated in epidemics. By some observers, however, it has been looked upon as a malarious form of yellow fever.

Further outbreaks occurred in Ireland and Scotland in 1847–48, and the disease now appeared in different English towns, including London, Croydon, Liverpool, and Manchester. Between 1868–73 it was again prevalent in Scotland and England, the earliest cases in London being 'chiefly in a quarter inhabited by Irish and by poor Jewish emigrants from Poland' (Hirsch).

Outside Britain the disease seems first to have been observed in Russia, where it apparently occurred in single epidemics in 1833 and 1840, and on an extensive and widespread scale in 1863–64. In 1878–79 it broke out again among the Russian troops in Bulgaria. It was observed in Germany in 1847, and was widely diffused in that country in 1868, appearing again in 1871–72 and 1878–79. There are a few references to the disease in the Scandinavian kingdoms and Belgium, but apparently none as regards France, Switzerland, Italy, Spain, and Portugal. Similarly, the Australian colonies seem to have been so far exempt. The disease has, however, occurred in North America, India, China, and Egypt.

Mortality.—In Table IV. will be found the deaths recorded in England and Wales as having resulted from relapsing fever in each of the years from 1869 to 1890, the deaths being given for males and females separately. From this table it will be seen that the mortality from relapsing fever has during the period in question always been insignificant, even in epidemic

TABLE IV.—*Showing the Deaths, at all Ages, of Males and Females registered in England and Wales in the Years* 1869–1891 *as due to Relapsing Fever.*

Year	Males	Females	Total	Year	Males	Females	Total
1869	72	67	139	1881	6	9	15
1870	301	327	628	1882	4	9	13
1871	155	172	327	1883	7	9	16
1872	25	38	63	1884	6	6	12
1873	21	20	41	1885	5	5	10
1874	13	10	23	1886	3	4	7
1875	25	27	52	1887	3	6	9
1876	17	13	30	1888	4	4	8
1877	22	13	35	1889	1	2	3
1878	11	14	25	1890	2	1	3
1879	10	12	22	1891	4	7	11
1880	9	12	21				

years, as compared with that of typhus, enteric, and some of the other fevers. It will also be observed that it has diminished during successive years in a manner similar to that of typhus.

Fatality.—The fatality or case mortality of relapsing fever is low. Dr. Grimshaw gives it as from 1·2 to 2 per cent. for London, and as up to 4 and 4·5 per cent. for other places, the average rate being about 4 per cent. Murchison's statistics give the rate for Great Britain as 4·03 per cent.

Influence of Sex.—More males appear to be attacked than females. This Murchison considered due to the fact of there being a larger proportion of

males among the class most prone to the disease, viz. the vagrant class. But although more males appear to be attacked, more females die of the disease, so that the case mortality among the latter, at all ages taken together, is higher than that among the former. According to Murchison's London Fever Hospital figures, the case mortality was 1·64 per cent. for males and 2·15 for females; 'but this result is attributable to a larger proportion of the males being under thirty years of age. Under fifty the mortality was greater among females, but above fifty it was much greater among males.'

And Age.—As with typhus and enteric fever, so with relapsing fever, the largest number of attacks occur in the age period fifteen to twenty years. But the subsequent fall, especially during the years twenty-five to forty-four, is less sudden than in the case of enteric fever, and, in this respect, more resembles that of typhus.

The following table, which is compiled from three tables given by Murchison, shows the percentage of attack at each period of life by each of these three diseases. The percentages are based upon 18,138 cases of typhus, 2,111 of relapsing, and 5,911 of enteric fever.

TABLE V.

Age	Percentage at each period of life, of attacks at all ages			Age	Percentage at each period of life, of attacks at all ages		
	Typhus	Relapsing	Enteric		Typhus	Relapsing	Enteric
Under 5 years	1·29	1·84	·98	From 50 to 54	4·35	3·78	·60
From 5 to 9	6·59	5·96	9·44	,, 55 to 59	2·42	1·84	·33
,, 10 to 14	12·06	11·08	18·16	,, 60 to 64	2·20	2·55	·33
,, 15 to 19	16·16	19·13	26·86	,, 65 to 69	1·03	·56	·08
,, 20 to 24	13·23	16·81	19·69	,, 70 to 74	·46	·23	·00
,, 25 to 29	9·52	9·80	10·15	,, 75 to 79	·17	·04	·03
,, 30 to 34	8·36	8·43	5·36	Above 80	·01	·00	·00
,, 35 to 39	8·03	6·82	3·40	Age not specified	·00	·00	1·30
,, 40 to 44	8·30	6·72	2·09				
,, 45 to 49	5·72	4·26	1·08	——	99·90	99·85	99·88

The fatality of relapsing fever is very low during the early years of life, but, like that of typhus, markedly increases with advancing years. Murchison gives the following London Fever Hospital figures:—

Under 30 years,	1,366	cases,	7	deaths, or	·51	per cent.
Above 30	,,	745	,,	32	,,	4·29	,,
,, 50	,,	191	,,	18	,,	9·42	,,
,, 60	,,	72	,,	9	,,	12·50	,,

Influence of Season.—Relapsing fever seems to exhibit a considerable independence of season. On the whole, perhaps its attacks are somewhat more common in the winter than during the warmer periods of the year. The mortality also appears to be greatest in the winter.

Cause and Modes of Dissemination.—The phenomena of relapsing fever are believed to be bound up with the presence in the blood of a particular spirillum (see page 171).

The predisposing causes of relapsing fever appear to be identical with those of typhus, viz. overcrowding, filth, and starvation. Murchison maintained that starvation was the main factor, relapsing fever differing somewhat from typhus in this respect; but the experiences of other observers throw great doubt upon this, for in some notable epidemics starvation, according to the records, certainly cannot be said to have been a conspicuous etiological factor. Rather it seems that overcrowding, and all which that entails, is the important element, though, no doubt, starvation, fatigue, and the like are powerful

contributory causes when present. As regards the influence of overcrowding there seems no doubt.

When once established, relapsing fever is highly infectious. 'The mode of communication,' Murchison remarks, 'is probably the same as in typhus —that is to say, the poison is conveyed through the air, or by fomites, from the sick to the healthy, and actual contact is not necessary.'

With regard to the distance to which the poison will travel through the atmosphere, Murchison further says: 'It is only they who are in close communication with the sick, or who visit, or reside in, their badly ventilated dwellings that suffer. With free ventilation the disease almost ceases to be communicable.' Medical men and nurses are, as in typhus, particularly liable to contract the disease, and laundresses also frequently contract it from washing the clothes of patients.

There are no grounds for thinking that soil, or topographical or geological circumstances, have any influence over the rise and spread of the disease.

Period of Incubation.—More exact data are wanted with respect to the period of incubation. Murchison considered that, on the whole, it was shorter than that of typhus. Like that of typhus, however, it seems to be subject to considerable variation. The Silesian physicians gave it as from fourteen to twenty-one days, and Lebert as from three to seven days. Among the twelve cases in which Murchison considered that he definitely fixed it, there were variations from 'attack immediate on exposure' up to fourteen days.

Protection.—Relapsing fever appears to afford comparatively little protection against subsequent attack. Certainly, second attacks are of much more common occurrence than in the case of typhus.

Relation to other Diseases.—Relapsing fever has generally been observed to occur in epidemic association with typhus, and the most usual course of events in these mixed epidemics has been, as Murchison shows, that 'the proportion of relapsing cases has been greater at the commencement than towards the close of the epidemic, and with the advance of the epidemic typhus has taken the place of relapsing fever.'

The commonly accepted explanation of the frequent association of these two diseases is that their *predisposing* causes are similar, the diseases themselves being held to be specifically distinct, and therefore dependent upon different *essential* causes.

The belief in the separate nature of the two diseases appears to be based mainly upon the following considerations: (*a*) that they present marked clinical differences; (*b*) that the one disease does not give rise to the other; (*c*) that one does not protect against the other; (*d*) that Obermeier's spirillum is not observed in the blood of typhus patients, but is always present in that of relapsing fever patients during the relapse.

These arguments, if all well founded, must reasonably be allowed considerable weight. Nevertheless, as elsewhere pointed out, it is by no means impossible that diseases possessing relatively specific characters may yet have some genetic relation to one another, and thus it must not be too hastily concluded on the ground of clinical, or even apparent bacteriological, differences that such diseases are entirely unrelated.

Looked at from this standpoint, it is difficult to avoid being struck with the similarity, as regards epidemiological features, of these two diseases. They admittedly occur under much the same conditions, and relapsing fever is usually associated with typhus. They both seem to be entirely independent of soil, and largely independent of season. In their modes of dissemination they are strikingly similar—so similar, indeed, that Murchison, a firm sup-

porter of the 'separate' doctrine, says of the behaviour of the relapsing fever infection that 'the remarks made on this subject under the head of Typhus apply with equal force to relapsing fever.' Considering the peculiarities of the typhus infection, this is a suggestive circumstance. The diseases also resemble each other in the marked increase of their fatality with advancing years of life; and there is, too, a general resemblance between their age incidence in the matter of attack. Their latent period seems to be similarly variable. The geographical distribution of *recognised* relapsing fever, it is true, is less wide than that of typhus; but, as Murchison remarks, it is 'much wider than it was once supposed to be.' Moreover, relapsing fever was first recognised, and has been most conspicuously present, in countries especially prone to typhus, notably Ireland and Russia. Lastly, it seems doubtful whether the arguments mentioned above as forming the basis of the doctrine of the separateness of these two diseases are all founded upon fact.

The assumption that the one disease does not give rise to the other appears to be based on the statement that, during mixed epidemics, cases of both kinds are not found coming from the same households. But it is pointed out by Fagge that, according to Murchison's later records, this is not strictly true. Then, again, the great frequency with which mixed epidemics have been observed to consist mainly at the outset of relapsing fever and later of typhus may possibly be indication of 'progressive development.' Even, however, if both these positions should be untenable, it may still be that the two diseases are evolution varieties of a common stock, but breeding true, and consequently each producing only its own kind.

As regards the statement that the diseases do not protect against each other there seems also some doubt. It is true that well-authenticated cases of persons having suffered from both these diseases have been recorded by different observers. But such cases do not prove the proposition in question. Second attacks of both typhus and relapsing fever are known to occur, and protection is a matter of degree. On the other hand, the late Dr. J. C. Steele observed 'that in the epidemic of 1848 persons who had previously suffered from typhus were not attacked by relapsing fever; and the same thing is said to have been noticed recently in epidemics at St. Petersburg and at Breslau, . . . while Lebert draws attention to the fact that in his cases of typhus, among those who had already passed through relapsing fever, the mortality was only half as great as in the population generally. Do these facts,' it is very naturally asked, 'indicate that after all there is between the two diseases some connection the nature of which is not at present understood?'[1]

INFLUENZA

Synon.: *Epidemic Catarrh.* Fr. *La Grippe*; Ger. *Influenza*; It. *Influenza.*

History and Distribution.—When influenza reappeared in England at the close of the year 1889 there was a marked tendency among the public, and even the medical profession, towards the view that we had amongst us some entirely new scourge—so unfamiliar had we become in this country with the character of the malady. Yet, curiously, there is perhaps no disease which has so frequently swept over the face of the globe in pandemic waves.

Zuelzer, in his article on influenza in Ziemssen's 'Cyclopædia,' expresses

[1] Fagge, *op. cit.*, i. 154.

the opinion that that disease can only with certainty be traced back to the beginning of the sixteenth century, though he points out that epidemics of catarrhal fever which may have been influenza are recorded as early as the ninth century. Hirsch,[1] on the other hand, considers that 'the disease may be followed into the remotest periods from which we have any epidemiological record at all;' and in a paper read before the Society of Medical Officers of Health in February 1890, Dr. W. H. Clemow[2] referred to various interesting early epidemics dating back from the year 1481 to the time of Hippocrates, some of which would certainly seem to have been influenza. Ignoring earlier records, which were thought too deficient in detail to be of much use for epidemiological purposes, though 'they certainly relate to influenza,' Hirsch gives a long list of epidemics extending over the period from 1173 to 1875, and affecting at one or another time every quarter of the globe.

But the first extensive pandemic of which we have reliable information seems to have occurred in the year 1510, and Zuelzer remarks that with that year 'begins a series of epidemics, the wide distribution of which has been reached by no other acute infectious disease.'

The chief epidemics in this country during the present century occurred in the years 1803, 1833, 1837–38, and 1847–48. But in addition to these major epidemics, Dr. Parsons, in his report to the Local Government Board on the influenza epidemic of 1889–90, points out that in the earlier years which followed the major epidemics of 1837 and 1848 minor periodical recurrences at intervals of three or four years may be traced in the death returns of the Registrar-General down to the year 1858.[3] Whether the lesser prevalences should be regarded as the periodical flickerings of the dying virus left in this country by the larger epidemics, or whether they should be considered in connection with the prevalence of the disease in other countries during the same years, is doubtful, for it seems, according to Hirsch, that influenza was epidemic in Germany in 1841; in Germany, France, Russia, and North America in 1843–44; generally over the western and eastern hemispheres in 1850–51; generally over Europe, including the Faröe Islands and Iceland, in 1855–56; and generally again over the western and eastern hemispheres in 1857–58. Since that time influenza is recorded by Hirsch as having occurred in Australia, North America, Bermudas, Netherlands, Cape of Good Hope, and Iceland between 1860 and 1862; New Caledonia, France, California, and Switzerland in 1863–64; France, England, Mauritius, Germany, and Belgium, 1866–67; Turkey in 1868; universally over North America in 1873, and widely over Europe and North America in 1874–75. But notwithstanding this influenza prevalence in other countries, it would appear tolerably clear from the Registrar-General's returns that England enjoyed a comparative immunity from the disease for a number of years prior to 1889. The present epidemic, of which, during the past three winters, we have had such a painful experience, would, however, seem to be one of the most severe and widespread ever recorded. Its original progress over the globe was also more rapid than that of previous epidemics.

Geographical Distribution and Influence of Race, Climate, and Season.—A glance at the history of this disease, whether it be at the general history or the history of this latest epidemic, shows influenza to have a world-wide distribution, and consequently to affect persons of all nationalities. Climate, season, and weather also appear to have no controlling influence over its

[1] *Op. cit.*, i. 7.

[2] 'Epidemic Influenza,' by Dr. W. H. Clemow, *Public Health*, April 1890.

[3] See Table VI., p. 285.

spread, and Dr. Parsons points out, with respect to the 1890 outbreak, that, like former epidemics, it 'prevailed nearly at the same time at places in the north and south hemispheres, i.e. in opposite seasons of the year. It has prevailed in the cold of Russia and the heat of India; in the moist climate of the British Isles and the dry air of Egypt. In Spain its advent was preceded by a month of cold, dry, frosty weather, and at New York by the mildest and moistest season on record.' Speaking of former experience, Hirsch says that, in its origin, influenza is independent 'of the seasons and of the influences of the *weather*; and it is in that respect that it is marked off most essentially and most decidedly from epidemic bronchial catarrh.' Zuelzer expresses the same view. Telluric influences appear also without notable effect upon influenza.

As to the original home of the disease nothing is known. But in the northern hemisphere influenza epidemics have exhibited a tendency to travel from east to west. This is stated by Zuelzer, Watson, and others; and Hirsch, although he appears to consider that such a 'progress of the disease in a definite line from east to west' has been exaggerated, admits that it cannot be denied that 'some pandemics, regarded as a whole, do afford evidence of the sickness travelling in the alleged direction, from east to west.' The present epidemic certainly followed this course.

Periodicity.—Of any regular and sustained periodicity history seems to afford no evidence, though the annual recurrences of the disease during the last three or four years might perhaps point to an annual developmental cycle of the poison. The apparent tendency of the disease on previous occasions to recur at intervals of three or four years has already been referred to.

Variation in Type.—Influenza epidemics differ in type from time to time, and similar differences are commonly observed among different cases of the same epidemic. Thus the prominence of ordinary catarrhal symptoms may vary considerably. In the present epidemic such symptoms have been less conspicuous than on former occasions, though it has been especially pointed out by observers of previous epidemics that catarrh, in the ordinary sense, may be entirely absent. Again, the gastro-intestinal, pulmonary, and nervous symptoms may each be more or less prominent at different times, though all, and especially the last, seem to be present in every epidemic.

Different epidemics have, moreover, preserved the same general characters, such as rapidity of dissemination, general independence of climatic, seasonal, age, and sex influences, relative suddenness of onset as regards attack, and low case mortality.

It has been stated above that when the present epidemic appeared in 1889 many hesitated to regard it as influenza. This, however, was largely due to the erroneous conception of influenza which prevailed as a result of the improper application of the term 'influenza' during non-epidemic periods to ordinary catarrh. But, as Dr. Parsons remarks, 'few persons who have read the history of the previous epidemics of influenza, and compared it with that of the epidemic of 1889–90, can doubt that the disease with which we have been visited is the same as that which has prevailed so extensively in former periods.' Certainly influenza presents certain points of similarity to dengue, and some persons were at first not unnaturally inclined to believe the present epidemic to be one of that disease. Dengue, however, is essentially a disease of hot climates and seasons, is seldom fatal, is unattended with pulmonary complications, almost always presents a rash, and is frequently followed by profuse desquamation. Other clinical distinctions also seem to exist between the two.

Mortality.—In Table VI., page 285, will be found year by year the total

TABLE VI.—*Showing year by year the Deaths registered in England as due to Influenza, the corresponding Influenza Death-rate per Million of the Population, and also the Death-rates from diseases of the Respiratory and Circulatory Organs.*

England and Wales				
Year	Total deaths from influenza	Death-rates per million from		
		Influenza	Diseases of the respiratory organs	Diseases of the circulatory system
1838	806	55	—	—
1839	887	59	—	—
1840	1,030	67	—	—
1841	1,659	106	—	—
1842	833	53	—	—
1847	4,881	285	2,980	580
1848	7,963	460	2,587	556
1849	1,611	92	2,536	618
1850	1,380	78	2,184	637
1851	2,152	120	2,705	657
1852	1,359	76	2,646	699
1853	1,789	99	3,118	760
1854	1,061	58	2,856	734
1855	3,568	193	3,439	786
1856	1,029	55	2,812	726
1857	1,393	73	3,057	775
1858	1,794	93	3,399	852
1859	1,112	57	3,069	879
1860	1,130	58	3,484	956
1861	746	38	3,233	909
1862	915	45	3,358	930
1863	919	45	3,308	959
1864	804	39	3,363	1,089
1865	596	29	3,291	1,078
1866	651	31	3,592	1,059
1867	607	29	3,312	1,076
1868	306	14	2,847	1,049
1869	703	32	3,594	1,144
1870	615	28	3,626	1,151
1871	348	15	3,569	1,177
1872	278	12	3,147	1,193
1873	266	11	3,632	1,244
1874	245	10	3,797	1,289
1875	449	19	4,282	1,381
1876	203	8	3,656	1,333
1877	205	8	3,547	1,413
1878	195	8	3,830	1,445
1879	266	11	4,332	1,513
1880	171	7	3,614	1,372
1881	99	4	3,428	1,369
1882	90	3	3,565	1,376
1883	107	4	3,675	1,473
1884	72	3	3,342	1,506
1885	138	5	3,737	1,613
1886	83	3	3,641	1,647
1887	85	3	3,626	1,666
1888	92	3	3,502	1,695
1889	55	2	3,309	1,664
1890	4,523	157	4,120	1,757
1891	16,686	572	4,474	1,826

influenza deaths recorded by the Registrar-General in England and Wales during registration times, and the corresponding death-rate per million of the population. The death-rates per million living are also given for diseases of the respiratory organs and diseases of the circulatory system. It will thus be seen that a rise in the deaths from influenza has generally been attended by a rise in the mortality from lung disease and sometimes heart disease. During the years 1890 and 1891 this is especially con-

spicuous, but it may be traced elsewhere in the table.[1] The mortality attributable to influenza must therefore not be measured solely by the deaths registered as due to that cause, but the indirect effect of the malady as expressed in the increased mortality from certain other causes must be taken into account. Accordingly the Registrar-General estimates that, during 1890, 'the total number of deaths due directly or indirectly to the epidemic influenza was not merely 4,523, but 27,074, or 941 per million living.'

Case Mortality.—The actual mortality from influenza, in such an epidemic as we are now passing through, is therefore a sufficiently serious matter. The fatality, however, i.e. the proportion of deaths to attacks, is low, and this seems to have been a constant characteristic of influenza. Dr. Parsons found that among the in-patients treated in eight large London hospitals, from which during the year 1890 he obtained returns, the fatality was 34·5 per 1,000 cases. Among the total patients at the same hospitals, 'in' and 'out,' amounting in number to 5,516, it was only 1·6 per 1,000 cases. In the Army, where the patients' ages and circumstances are favourable for recovery, there were 9 deaths among 8,103 cases, or 1·1 per 1,000. In the more recent epidemics the case mortality seems to have been greater.

Influence of Age and Sex.—The mortality from epidemic influenza is greatest during the middle and later periods of life, and in this respect there is a marked contrast between that disease and what in non-epidemic periods is loosely called influenza, for in the latter condition the mortality is mainly among children and old people. The difference in the mortality of these two maladies at different ages is well shown in the following table from Dr. Parsons' report:—

TABLE VII.—*Proportion at the several Ages to* 100 *Deaths from Influenza at all Ages, in London.*

Period	Percentage at several ages							
	Under 1	1–5	5–20	20–40	40–60	60–80	80 and above	Total
1876–89	32·8	16·0	3·4	3·4	10·0	26·9	7·5	100
First quarter, 1890 . .	5·2	4·3	4·7	24·7	36·2	22·4	2·5	100

In the epidemic of 1847–48 the age mortality differed somewhat from that during the first quarter of 1890, in that the greatest proportion of deaths occurred in the age period 60–80, instead of 40–60, as shown in the table. But it also differed from that of the influenza of non-epidemic times 'in not showing the high proportion of deaths in early childhood.' As regards liability to attack, Zuelzer, referring, of course, to previous epidemics, says: 'Everybody is agreed in stating that epidemics attack the population without distinction of age, sex, constitution, or condition. . . .' That children cannot be regarded as in any special degree exempt is proved by the large percentage attacked in different schools and training ships.

Cases of supposed influenza in new-born infants have also been reported.

With respect to the incidence of the disease upon the different sexes, it appears from the Registrar-General's reports that in the epidemic years 1847–48 there were 6,819 deaths of females registered as influenza, and 6,025 of males. Dr. Peacock attributed the excess of female deaths to the

[1] It is somewhat masked by the steady increase, from other causes, in the mortality from heart and lung diseases. It will best be appreciated by comparing the mortality from these diseases in influenza years with that of the years immediately succeeding them.

fact of the disease being mostly fatal to persons of advanced age, and it has already been seen that the greatest proportion of the deaths in that epidemic was at the age period 60–80. In the year 1890 we see from the Registrar-General's annual report that of the 4,523 influenza deaths registered, 2,108 were of females and 2,415 of males. The mortality for that period was therefore greatest among males; but the greatest proportion of the 1890 influenza deaths occurred in the age group 40–60; and further, the proportion of deaths in the 20–40 period was, for London, greater than that among persons in the 60–80 group. This, therefore, would seem to support Dr. Peacock's view. Moreover, in 1890 the female mortality was greatest at all ages over sixty-five, but at all other age groups, except one or two of the earlier ones, where the numbers were small, the mortality was higher among males—probably as a result of greater exposure to infection and cold.

On the whole, then, it would seem that the mortality from influenza is little influenced by sex, except perhaps in connection with differences as regards occupation. Neither do the sexes seem to differ in liability to attack if similarly circumstanced. The number of male and female clerks attacked in the Post Office in 1890 were in proportion to the numbers of each sex.

Protection.—Influenza is frequently said to confer no protection against future attack. Certainly, a large number of persons suffer a second and third time under circumstances which point to a reinfection. At the same time, it must be borne in mind that the fact of many persons undergoing second or third attacks does not prove that other persons have not been protected, or even that the persons suffering from more than one attack have not been protected for a period during the interval, except in cases in which the second attack follows closely upon the first; and cases of the latter sort are open to the suspicion of being the result of relapse or recrudescence.

Cause and Dissemination.—Various hypotheses have been framed as to the cause of influenza, but the most probable one, in view of recently acquired knowledge, both with respect to the origin of other diseases and the behaviour of influenza, is that it depends upon a micro-organism. Moreover, there would appear good grounds for thinking that a particular bacillus has been shown to stand in causal relation to that disease by Pfeiffer and Kitasato.

But, however that may be, it seems now established that influenza is an eminently infectious complaint—a fact which, although often maintained by individual authorities in the past, has been frequently denied by others. It is impossible here to enter into the evidence on this point in detail. The following is, however, the summary of Dr. Parsons' conclusions on this matter:—

'In view of the circumstances—1. That the progress of the epidemic was contrary to the prevailing winds, and that it was independent of season or any particular kind of weather; 2. That it has not been shown to have travelled faster than human beings could travel; 3. That it has not occurred among persons placed under circumstances precluding its communication by human agency; 4. That, as a general rule, in each country it has appeared first in the capital, or the ports of entry, or the frontier towns in communication with countries previously invaded, and that the towns, as a rule, have been affected earlier than country places; 5. That neighbouring communities have in certain instances been affected only at considerably different dates; 6. That many instances are recorded of the disease having been introduced into a district, and spread to persons in contact with the patient, and sometimes afterwards to others; 7. That persons brought much into contact with others—e.g. people going daily to business in towns, have generally

been the first to suffer; their households, and people locally employed, being affected later; 8. That in public services and establishments persons employed together in large numbers in enclosed spaces have suffered in larger proportion than those employed few together, or in the open air; 9. That in institutions in which the inmates are brought much into association the epidemic has more quickly attained its height, has prevailed more extensively, and been sooner over than in those in which the inmates are more secluded from one another; I am of opinion that the epidemic has been propagated mainly, perhaps entirely, by human intercourse; though not in every case necessarily from a person obviously suffering from the disease. I see no sufficient ground for believing in that world-wide spread by atmospheric agencies which has been so generally assumed. I do not say that the contagion once imported into a locality may not propagate itself outside the human body in such media as damp ground or air contaminated with organic exhalations, but the fact of adjoining communities suffering at different dates seems opposed to the notion of the poison travelling far through the air. I do not find sufficient evidence that the recent epidemic has anywhere commenced suddenly with a large number of simultaneous cases, unpreceded by any previous ones, and I think that the rapidity with which influenza develops into an epidemic may be accounted for by its short period of incubation, by the comparatively general susceptibility to the disease, and by the existence of numerous slight and unrecognised cases. I do not, however, wish to exclude the possibility that the specific germ of the disease may multiply in appropriate media, e.g. in damp, organically polluted confined air, outside the human body.' [1]

The question, too, of the possible communicability of influenza from animals to man, or *vice versâ*, has to be considered, and it is suggestive to find among the records of influenza epidemics from the earliest times reference to concurrent illness among domestic animals, notably horses, dogs, and cats.

With respect to this subject Hirsch says: 'Even in the oldest epidemiological records, there are indications of these coincidences, both as regards time and place, as well as of the identity or at least similarity of the form of disease; and the number of these observations is so remarkably large that the suggestion of an etiological and perhaps also pathological connection between the epidemics on the one hand, and those epizootics on the other, may be regarded as provisionally proved, although it ought not at the same time to be left out of sight that the notion of "horse influenza" has remained to the present day a somewhat vague one with veterinary surgeons, and that very various diseased processes appear to have been included therein.' Sir Thomas Watson says: 'It has been observed also that shortly before, or during, or soon after, the prevalence of these epidemic catarrhs epizootic diseases have raged; various species of brutes, and of birds, have been extensively affected with sickness.'

Certainly influenza or 'pink eye' was prevalent among horses in different parts of England either before, during, or after the epidemic of 1890; but as regards this epidemic Dr. Parsons is of opinion that the origin of influenza in the human subject from a similar disease in the horse is rendered improbable by the following considerations:—

1. That 'influenza' among horses has frequently prevailed at times and places when there has been no human epidemic; 2. That in the late epidemic persons having to do with horses were not observed to be specially

[1] See also the evidence brought forward by Dr. Sisley on 'Epidemic Influenza.'

or earliest affected; and that cases of apparent transmission of the disease from the horse to man were of somewhat rare occurrence; 3. That in many places where the late influenza epidemic prevailed (e.g. at Newmarket), the absence of any similar disease among horses was affirmed.

Dr. Parsons, however, reports that in many places where influenza was prevalent pet dogs, cats, and caged birds, especially those living indoors, were noticed to concurrently suffer from similar symptoms.

The likelihood of the microbe having ability to live for a considerable period outside the body seems supported by the fact that after an epidemic the disease tends for a time to recur at intervals in the same locality, as we have unfortunately experienced of late. In reference to this, Sir Thomas Watson says: 'The locality does not appear to be thoroughly cleared of the poison for some time; or perhaps a more cautious statement of the fact would be, that the disorder generally shows itself again in succeeding years, but in a milder and less general form.'

Periods of Incubation and Infectiveness.—It is not easy to fix the period of incubation of a disease which spreads so rapidly as influenza, owing to the difficulty of eliminating the fallacy of multiple exposure. The evidence collected however, makes it certain that the period is commonly a short one, and the most usual time would seem to be two to three days, though it may be longer or shorter. Influenza is infectious quite in the early stage, and certainly as long as the eighth day, and perhaps longer.

WHOOPING COUGH

Synon.: *Pertussis.* Fr. *Coqueluche*; Ger. *Keuchhusten*; It. *Tosse Convulsa.*

History and Distribution.—There seems no sufficient evidence that whooping cough is a disease of great antiquity. Hirsch gives an epidemic in Paris in the year 1578 as the first reliable record of the disease. In England whooping cough was referred to in the seventeenth century by Sydenham and Willis.

At the present day the disease has practically a world-wide distribution, but is more constantly prevalent and more severe in temperate climates than in tropical regions. So far, however, there is no evidence that race influences liability to attack, for there are accounts from India, Egypt, and other countries, of coloured children suffering equally with Europeans.

But, although the disease appears, at one or another time, to have visited most parts of the world, its introduction to some countries, as Australia and New Zealand, appears to have occurred during the present century, as a result of importation. Similarly, in other localities having but little communication with the rest of the world, as Iceland and the Faröe Islands, it has only occurred at apparently rare intervals when introduced from without.

Mortality.—In the following table will be found the mortality recorded from this disease in England and Wales from 1838 to 1891.

A glance at this table will show that the mortality from whooping cough in this country is very considerable indeed; of late years this disease has destroyed more children than any of the other so-called zymotic diseases, with the exception of diarrhœa.

Case Mortality.—The case mortality of whooping cough is not high, and is sometimes stated at about 5 per cent. But in addition to the number of

children who die from this disease, a very considerable number are more or less permanently injured by it.

Influence of Season.—The effect of season upon whooping-cough mortality

TABLE VIII.—*Showing year by year the Deaths registered in England and Wales from Whooping Cough, and the corresponding Death-rates per Million of the Population.*

England and Wales							
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1838	9,107	596		1867	11,873	548	
1839	8,165	526		1868	9,223	419	545
1840	6,132	389	504	1869	10,966	493	
1841	8,099	508		1870	11,901	529	
1842	8,091	502		1871	10,360	455	
1847[1]	9,260	540		1872	13,806	596	
1848	6,862	394	480	1873	9,612	411	499
1849	9,622	548		1874	10,362	437	
1850	7,770	437		1875	14,280	594	
1851	7,905	440		1876	10,556	432	
1852	8,022	440		1877	11,358	460	
1853	11,200	609	511	1878	17,784	710	527
1854	9,770	525		1879	12,752	503	
1855	10,185	541		1880	13,662	530	
1856	9,225	483		1881	10,830	415	
1857	10,138	526		1882	15,259	579	
1858	11,648	598	498	1883	10,471	393	459
1859	8,976	456		1884	11,476	425	
1860	8,555	429		1885	13,106	481	
1861	12,309	612		1886	12,936	470	
1862	12,272	602		1887	11,251	404	
1863	11,275	547	516	1888	12,287	436	444
1864	8,570	409		1889	12,225	430	
1865	8,647	409		1890	13,756	478	
1866	15,764	736		1891	13,612	468	

in London will be seen from the curve on the next page. This curve shows the London mortality from whooping cough is at its minimum in September, rising steadily through November and December to its maximum in April, thenceforward steadily declining through the warmer months. It is usually said that the prevalence of whooping cough in this country, like the mortality, is greatest about the months of March and April. It will be noted that this curve is almost exactly the reverse of that for scarlet fever.

The relation to season thus indicated does not seem to hold for all other countries. According to Hirsch, in Sweden, for instance, both the prevalence and mortality of the disease appear to be greater in the summer and autumn than in the winter. As regards the effect of weather, Dr. Goodhart[2] remarks: 'Atmospheric changes have a most important bearing upon pertussis. It has been repeatedly noticed in the whooping-cough ward at the Evelina Hospital that the children are worse, even when otherwise doing well, when the wind turns cold or suddenly changes; and it is notorious that the disease runs a much less determined and persistent course in summer than in the colder seasons of the year.'

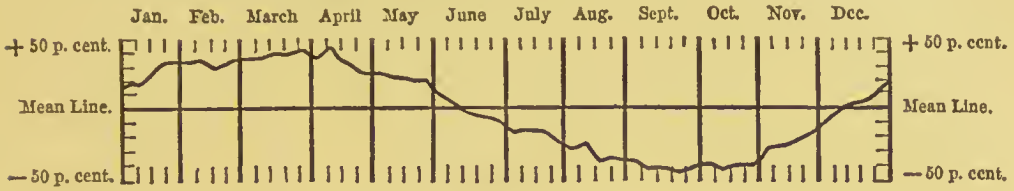
Influence of Age and Sex.—The mortality from whooping cough is decidedly greater during the first year of life than during any subsequent year.

[1] Deaths not abstracted by Registrar-General for years 1843–46.

[2] *Op. cit.*, p. 232.

Reference to the Registrar-General's annual reports will show that between 40 and 50 per cent. of the whooping-cough deaths at all ages occur within

Whooping Cough—for all Ages and both Sexes (Buchan and Mitchell)
(London Deaths from 1845 to 1874).



this period. During the second year the mortality is considerable, but it falls rapidly during subsequent years, and after the tenth year is insignificant.

Of the total deaths, over 90 per cent. at all ages occur during the first five years of life. The incidence of the disease as regards attack seems also to be mainly upon the earlier years of life, though no age is entirely exempt. Of 314 cases collected by Dr. Goodhart, 301 occurred in the first six years of life, sixty-two of these being in the first year. The mortality among females is greater at all age periods than among males. One attack of whooping cough usually confers immunity against a repetition of the disease.

Periods of Incubation and Infectiveness.—Period of incubation is stated by some observers to be from four to six or seven days, but it appears liable to variation. The late Dr. Murchison reported some cases which appear to be free from any likelihood of fallacy, in which the incubation period was apparently a fortnight.[1] Other more recent authorities have also given fourteen days as a common period. Whooping cough is highly infectious from the very commencement of the attack, before the whoop manifests itself, and remains infectious for some six or eight weeks, as a rule. Most authorities seem agreed that the infective period may be considered to have ceased at the end of six weeks from the commencement of whooping, provided all spasmodic cough has ceased.

Cause and Dissemination.—The epidemic and infectious character of this disease, together with the facts of its having an incubation period, and affording protection against a second attack, are circumstances which seem to justify the conclusion from analogy that it is of microbic origin, but at present no micro-organism has been shown to stand in causal relation to it. The disease is, so far as at present known, invariably spread by infection from case to case. Such infection, however, need not necessarily be direct, as the virus may be carried in clothing, &c. Owing to the early infectiveness of whooping cough, it is, like measles, largely spread by the attendance at schools, and other public gatherings, of children who are sickening for it, but who have not, so far, manifested the characteristic symptoms. There is no evidence that this disease is ever disseminated by the agency of water, milk, or domestic animals. Neither does it appear to have been shown to have any relation to any particular topographical or geological circumstances.

Relations to other Diseases.—Whooping cough is usually said to occur frequently in association with measles. It seems probable, however, that this association has been exaggerated, and as to the order of sequence in which these two diseases occur with respect to each other, there seems also difference of opinion. Dr. Goodhart remarks that 'when an appeal is made to statistics, the association appears to be less common than I had anticipated. Of 305 cases of pertussis of which I have notes, measles is only mentioned as recent in fourteen.' Dr. Goodhart also found that in all these cases the

[1] *Trans. Clin. Soc.*, xi. 1878.

measles preceded the whooping cough. Other observers, on the other hand, have described the whooping cough as preceding the measles. Hirsch found that in 495 epidemics of whooping cough, a coincidence with epidemic measles was noted ninety-four times. 'In fifty-eight of these the diseases occurred together; in eleven whooping cough preceded measles; and in twenty-five it came after.'

MUMPS

Synon.: *Parotitis; Cynanche Parotidea* (Cullen); *Parotitis Epidemica; The Branks.* Fr. *Les Oreillons*; Ger. *Ziegenpeter*; *Bauerwetzel*; It. *Parotitide.*

History and Geographical Distribution.—According to Hirsch, epidemic inflammation of the parotid gland is described 'in a masterly fashion' by Hippocrates, who records, among other things, the liability of the testicle to become inflamed during the course of the malady. The distinctness of epidemic from symptomatic parotitis seems also to have been clearly recognised by the early Greek and Roman writers generally, as well as by the mediæval practitioners. Evidently, then, mumps is a disease of considerable antiquity.

In the present day its geographical distribution is practically coextensive with the habitable globe. In its diffusion mumps is at times limited to particular areas, or particular sections of the community, such as the children at certain schools, or the occupants of particular barracks, workhouses, or prisons. But at other times its epidemicity is more marked and it extends over wide areas of country. Sometimes it displays a tendency to recur in a locality at short (as yearly) intervals, but as a rule a longer period elapses between successive epidemics.

Influence of Climate and Season.—That no particular climate will prevent the occurrence of mumps is evident from the records of epidemics of the disease in Iceland and Lapland on the one hand, and in India, Arabia, and the West Coast of Africa on the other—as well, of course, as throughout the countries in the temperate zone. Nevertheless, as a rule, mumps is decidedly most prevalent during the cold and wet seasons of the year.

Mortality.—The mortality from mumps is insignificant. From the Registrar-General's annual reports for the ten years 1881–90 it appears that among the entire population of England and Wales some eighty deaths only are annually egistered as due to this disease. Even this, however, probably overstates the actual mortality from mumps, for there can be little doubt that some of the deaths so registered are due to other maladies, notably diphtheria.

Fatality.—In view of the wide prevalence of the disease in this country, and of the few deaths ascribed thereto, it is clear that the fatality is insignificant.

Influence of Age and Sex.—Mumps is said to be most common in children from ten to fifteen years of age, but the greatest registered mortality is among the very young. Thus, of the 800 odd deaths from mumps recorded by the Registrar-General in England and Wales during the ten years above referred to, about 68 per cent. occurred during the first five years of life;[1] and

[1] It may be suspected that not a few of these deaths were in reality due to other causes, notably diphtheria.

during those years the deaths were most numerous in the first year, diminishing with each succeeding year of the lustrum. The minimum was reached in the age period 15–20 years, after which there was a slight increase. Deaths occurred at all the age periods up to, and including, the period eighty-five years and upwards.

As regards sex, the deaths were decidedly more numerous among males than females, consisting of 472 for the former sex and 365 for the latter. In only two of the years did the female mortality exceed that of the male, and in one they were equal. In the remaining seven years the male deaths preponderated. This is in accord with the view expressed by different observers, that the disease is more common among males than females.

Cause and Mode of Dissemination.—It may be inferred from analogy that mumps is, in all probability, a microbic affection. So far as is known, it spreads only by infection from case to case, and it would therefore seem likely that the micro-organism upon which it is here provisionally assumed to depend belongs to the class of obligate parasites. The infection is believed to be given off by the breath. No relation between mumps and any particular telluric conditions has been made out.

Period of Incubation.—The period of incubation seems most usually to be from a fortnight to three weeks. It is probably seldom much less than twelve days.

Relation to other Diseases.—Mumps has often been considered to have occurred in association with measles and with diphtheria, and sometimes, though less frequently, with scarlet fever. But whether there is any relationship, other than a casual one, between mumps and the diseases in question is doubtful. Hirsch considers the observations too few to justify a conclusion on the point. As regards diphtheria at least, belief in concurrence of mumps therewith has often no doubt been due to error of diagnosis.

Protection.—One attack of mumps usually confers immunity, but second attacks do sometimes occur. Dr. Pye-Smith mentions an instance which came under his observation in which 'a boy had mumps three times during his school life.'

DIPHTHERIA

Synon.: *Angina maligna; Cynanche maligna; Putrid sore-throat.* Fr. *Diphthérie*; Ger. *Diphtheritis*; It. *Difterite.*

History.—It is only possible here to indicate a few of the more salient points of the history of diphtheria. Certainly diphtheria, or a disease very nearly allied to it, may be traced back to antiquity; and although Hirsch appears doubtful whether it is referred to in the Talmud and the Hippocratic writings, as has been alleged, he states that 'in the writings of some of the later Greek physicians, particularly Aretæus and Aetius, we meet with descriptions of an affection of the throat as to the identity of which with angina maligna there can be hardly any doubt.' As regards the writings of the Arabians and mediæval physicians of the West, Hirsch considers reference by them to 'angina' as often of doubtful import, but thinks 'there are accounts of epidemic forms of sickness in those ages given by some of the chroniclers, which may perhaps be taken as relating to malignant sore-throat.' In England there seems to have been an epidemic of angina in

1389, which carried off a large number of children ;[1] and in the Rhine districts there was, according to Hirsch, prevalence of an ' unknown ' sickness in 1517, 'so that men's tongues and throats were covered as if with a fungus and turned white, and they were neither able nor inclined to eat or drink from pains in the head not unattended with pestilential fever.'

In following the history of this malady into more recent times, two circumstances attract the attention—viz. the considerable extension which has apparently occurred in the geographical distribution of malignant throat ailment—at least as regards its conspicuous prevalence—and the variations which such ailment has also exhibited from time to time in the matter of prevalence generally.

Considering these two points together, we find that angina maligna was more or less continuously prevalent in Spain from 1583 to 1618, and reappeared in various parts of the country throughout the remainder of the century. Besides Spain, it also, rather later in point of time, visited Portugal, Southern and Central Italy, and the adjacent islands. At the beginning of the eighteenth century it again broke out in Spain. In 1736 it seems, judging from the accounts of Noah Webster, to have been extensively prevalent in England and America. About 1745–50 the disease again occurred in Portugal and Italy ; and now also, for the first time, come reliable records of its epidemic prevalence in France (north-east) and Holland, also of further outbreaks in England (at Liskeard and other places in Cornwall). About 1752 it was prevalent in Switzerland, Germany, and New York, and in 1755 in Sweden. Still later in the century the disease was epidemic in Portugal, the North of France, Holland, Germany (Osnabrück), the Northern States of America and England (London and Chesham).

Then, except in the case of France, seems to have come a marked remission, and Hirsch remarks that ' as the eighteenth century was drawing to a close, Angina maligna retired into the background among epidemic diseases which then held the stage.' This remission apparently lasted for the first half of the present century, when the disease again appeared on an extensive scale, assuming ' the character of a true pandemic, a character which it has maintained to the present day.'

It must not, however, be concluded that, except in the case of France, the disease entirely disappeared during the first half of the present century. Sporadic cases and even small epidemics occurred from time to time in most of the countries which had been previously visited ; but that such outbreaks must have been on a much smaller scale than during the preceding century Hirsch infers both from the meagre writings on the subject and from the fact that when, about 1860, the disease again became widely prevalent, it was ' expressly affirmed by many observers in all sorts of places . . . that they had no knowledge of the disease when they first saw it.' As regards Great Britain, cases apparently occurred in Dublin, Glasgow, Edinburgh, Warwickshire, Surrey, and Kent during the earlier years of the century ; and from 1845 to 1856 many scattered cases occurred in London, Kent, Lincolnshire, Herefordshire, Staffordshire, Norfolk, Devon, and Cornwall. In 1849 there was a decided epidemic at Haverfordwest, and in 1855 one at Launceston.

France, it has already been said, was exempt from the general remission. Throughout the period in question epidemics of diphtheria were both extensive and frequent in that country, although for the most part confined to the North. In 1821 Bretonneau, who had had opportunities of studying epidemics of malignant angina at Tours (1818–21), first propounded his doctrine of the essential identity of various throat ailments characterised by what he

[1] Webster's *History of Epidemic and Pestilential Diseases*, Hartford, 1799, i. 143.

then regarded as a membranous inflammation. To this condition of the air-passages he gave the name of *diphthérite* (διφθέρα, skin). Later, however, upon concluding that the morbid process was not an inflammatory one, he adopted the name of *diphthérie*. In connection with the persistence of the disease in France, and as bearing upon the question of the transport of the virus by human agency, it is interesting to note that the French troops suffered from diphtheria in the Crimea.

Proceeding to the latter half of this century we find, according to Hirsch, that ' this new era in the history of Angina maligna begins for the larger part of Europe and North America at almost all points with the years 1857 and 1858, a little earlier in some countries than others. . . . ' The diffusion of the disease, too, seems to have been far wider than hitherto. As regards France, it was no longer confined to the North. In England, as will be seen from Table IX., the mortality was excessively high in 1858–59.

Beyond Europe and North America, the disease about this period also became more or less extensively prevalent in parts of India, China, Australia, the West Indies, Argentine Republic, Peru, and parts of South Africa, though it is true there are accounts of earlier outbreaks in several of these localities.

From the above historical sketch it would seem clear, even allowing for the imperfect records of earlier times, that this disease has vastly extended its epidemic area, and that, during what may be called its historic period, it has exhibited one decided remission and a subsequent sudden, and almost world-wide, increase in epidemicity. It is striking also to observe how tenaciously diphtheria has adhered to certain localities, and how often places which had been previously affected by it were among the first to be attacked in later epidemics. As regards the very general prevalence of diphtheria which set in between 1855 and 1860, no doubt increased means of communication may have had some influence; but it would seem unlikely that greater facilities for locomotion constituted the only, or even the main, cause. Moreover, this increase of epidemicity has to be considered along with the previous remission and the still earlier prevalence of the eighteenth century. Such variations would seem incapable of being explained solely by variations in social circumstances, whatever minor part such circumstances may have contributed to the larger result; and we appear driven to the conclusion that the main factors were such as were bound up with conditions proper to the virus itself. The tendency of the disease to reappear in localities previously affected might mean simply that such localities offered a specially favourable environment of some kind to the cultivation of the virus whenever introduced from without; or it might mean that the diphtheria micro-organism, having once been introduced to such localities, has not ceased to exist during the non-epidemic periods, but has simply either more or less died down, or, perhaps, living a saprophytic existence, largely lost for the time being its pathogenic function. Subsequently, upon the re-occurrence of suitable conditions, meteorological or other, it has taken on fresh activity, or acquired a renewal of pathogenic property.

Of the more modern diphtheria prevalence in England we have now a broad indication in mortality statistics. The diphtheria deaths have only been abstracted by the Registrar-General since the year 1855, up to which time the few that occurred were included with those of scarlet fever.

The registered mortality in England from this disease rose gradually during the years 1855, 1856, and 1857, from 20 to 82 per million, and then with a bound in 1858 and 1859, in which years it was as high as 339 and 517 per million respectively. In reference to the excessively high rates in these two years, however, it has to be noted that during those years, and

especially the former, scarlet fever was also widely prevalent, and some allowance should doubtless be made for confusion between the two diseases. Nevertheless, it has been seen above that at this time diphtheria was becoming conspicuous over a large part of the civilised world, and it is certain that it showed a marked increase in this country also. Shortly prior to the more general extension of epidemicity in England, diphtheria, as we have seen, had occurred in various parts of the country, and was supposed to have been imported from France. Such may have been the case, but it is worthy of note that Cornwall, in which county the disease had occurred in the previous century, was now one of the earliest invaded. In the year 1860 the mortality fell to 261 per million, i.e. just half what it had been during the preceding year. For the decennial period 1861–70 it further fell to 187, and in the succeeding decennium it still further fell to 121. During the period 1881–90, however, there has been a decided increase in diphtheria mortality, the rate rising to 162.

One important point in connection with the behaviour of diphtheria in this country requires particular attention. Until recently diphtheria, according to all authorities, was especially a disease of sparsely-populated localities, but one of the most striking characteristics of its modern behaviour is its conspicuous invasion of the towns. Considering first the case of the metropolis, it appears as regards the period 1861–70 that, while the diphtheria mortality rate per million living was, for England and Wales, 187, it was for London only 179. But in the next decennium the rates were 121 and 122 respectively, and in the decennium 1881–90 they were 163 for England and Wales, and 259 for London. Thus, during the later period, although there was a decided increase in diphtheria mortality in the country generally, this increase was relatively far greater in the metropolis. But this urban invasion is by no means limited to London, as Dr. Longstaff has shown in his paper on 'The Geographical Distribution of Diphtheria in England and Wales.' Taking the Registrar-General's mortality returns for the twenty-six years, 1855–80, and dividing these years into three periods, and by also dividing the various counties in England and Wales into dense, medium, and sparse areas, according to the relative density per acre of their population, Dr. Longstaff was able to show that although during the whole period the sparse districts suffered most heavily from diphtheria, yet in each successive period the diphtheria mortality of the towns, relatively to that of the rural districts, had become greater.

In this paper Dr. Longstaff, in a map, here reproduced by his permission, shows that 'the distribution of diphtheria is apparently *sui generis*; the mortality from the disease clearly is not regulated by the same causes as influence the general mortality.'[1] It also differs vastly from that of the other zymotic diseases, as, for instance, diarrhœa, measles, and especially scarlet fever. The latter disease is most prevalent in the mining and manufacturing districts, some of which have particularly low diphtheria rates. The contrast between the distribution of these two diseases has also been pointed out by Dr. Edgar Barnes.

Periodicity.—Hirsch considers that the history of diphtheria indicates a cyclical character in the epidemicity of the disease, though 'the several cycles have extended over periods of various length, many of them only a few years,

[1] During the period in question (1855–80), the greatest diphtheria mortality occurred in the North Riding of Yorkshire, Lincolnshire, Norfolk, and Sussex; and the next greatest in East York, Cambridgeshire, Hunts, Essex, Kent, Surrey, Warwick, Wales (except Glamorgan), and some adjoining counties. In striking contrast was the low mortality of Devon, Somerset, Lancashire, West York, and various midland counties. This is illustrated by Dr. Longstaff in a shaded map.

West, Newman lith.

and others lasting several decades.' Looked at broadly, it may be that the prevalence of the eighteenth century, the remission during the first half of the present century, and the subsequent renewal of pandemic prevalence now going on, are the manifestations of an inherent periodicity with a rise and fall extending over a number of years.

TABLE IX.—*Showing the Deaths registered as due to Diphtheria in England and Wales during the years* 1855–1890; *the Annual Death-rate per Million living for England and Wales during the same Years; and the Annual Diphtheria Death-rates per Million living for London during the Years* 1859–91.

England and Wales			London	England and Wales			London
Year	Total deaths	Death-rate per million living	Death-rate per million living	Year	Total deaths	Death-rate per million living	Death-rate per million living
1855	385	20	—	1874	3,560	150	122
1856	603	32	—	1875	3,415	142	167
1857	1,583	82	—	1876	3,151	129	109
1858	6,606	339	—	1877	2,731	111	88
1859	10,184	517	284	1878	3,498	140	155
1860	5,212	261	174	1879	3,053	120	155
1861	4,517	225	239	1880	2,810	109	144
1862	4,903	241	255	1881	3,153	121	172
1863	6,507	315	275	1882	3,992	152	222
1864	5,464	261	207	1883	4,218	158	244
1865	4,145	196	144	1884	5,020	186	241
1866	3,000	140	152	1885	4,471	164	227
1867	2,600	120	145	1886	4,098	149	212
1868	3,013	137	158	1887	4,443	160	235
1869	2,606	117	107	1888	4,815	171	319
1870	2,699	120	104	1889	5,368	189	390
1871	2,525	111	105	1890	5,150	179	331
1872	2,152	93	80	1891	5,036	173	340
1873	2,531	108	95				

Mortality.—In the above table will be found the total deaths from diphtheria registered in England and Wales annually from the year 1855 to 1891. The annual death-rates per million living are also given; and for the purpose of indicating the relative increase during late years in the mortality from this disease in London, as compared with that of the country generally, the annual death-rates for London have been added.

Case Mortality.—The case mortality of diphtheria varies so greatly that it is impossible to form any useful estimate on the subject. In well-pronounced diphtheria, and especially in certain epidemics, it is unquestionably high, but it is impossible to gauge at all the number of slighter cases of diphtheritic throat illness, which, as will be seen later, so frequently accompany or precede diphtheria epidemics.

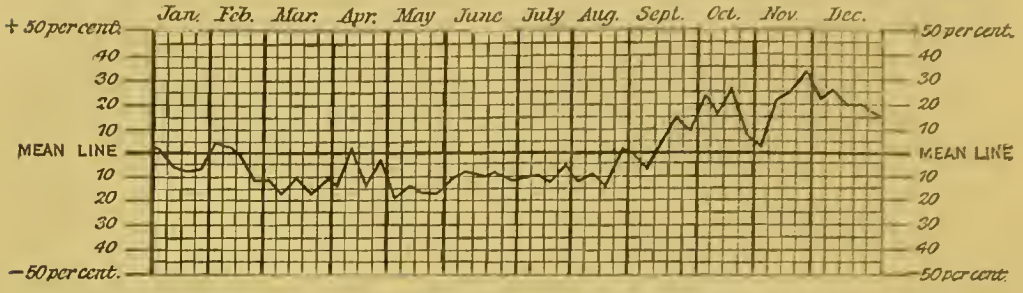
Influence of Climate and Seasons.—Diphtheria would seem to be capable of occurring in any climate, but it is far more common in temperate and cold regions than in the tropics. In this country the mortality from diphtheria is very regularly at its highest during the December quarter, and lowest during the summer months. This is shown for London in the curve (p. 298) published by the Registrar-General in his Annual Summary for 1890.

There is also evidence from notification returns that the prevalence of diphtheria, as well as the mortality, is greater during the autumn and winter than during the warmer period of the year.

The same general relation of diphtheria prevalence and mortality to the different seasons of the year would also seem to hold as regards other countries—at least in temperate climates. Hirsch, for instance, found that

the recorded diphtheria attacks in Sweden during the ten years 1861–70 were considerably more numerous in the winter quarters than in the summer.

Diphtheria—London Deaths for all Ages and both Sexes for Thirty Years, 1861–90.



He also found this true of the registered deaths (for differing periods of years) in a number of separate towns, including Berlin, St. Petersburg, Vienna, and Philadelphia. How far the influence of season upon diphtheria prevalence is direct, operating upon the activity of the diphtheria microbe, and how far it is indirect, and operates by increasing the susceptibility of individuals, is doubtful, but it appears probable that it acts in both these ways. There can be little doubt that anything which tends to damage the integrity of the mucous lining of the throat, such as ordinary catarrhs, would so far predispose to attack by diphtheria, given the presence of the efficient cause of that disease; and it is also possible that the influence of cold and damp may increase the general susceptibility of the body to infection. Nevertheless, it seems in a high degree probable that the activity of the diphtheria organism is also largely controlled by season.

Influence of Race.—The balance of present evidence would seem to be to the effect that there is no racial immunity to this disease.

Influence of Sex.—For the first two years of life the mortality from diphtheria is greater among males than among females. But from the third year until about the thirty-fifth it is greater among females. After that period the male mortality seems again to be, if anything, slightly in excess of the female.

The excess of female mortality, at certain ages, at least, is no doubt largely due to greater exposure to infection—that is, to the closer and more continuous contact with the sick to which females are exposed as compared with males; but Dr. Arthur Downes [1] has pointed to the very early ages at which the excess of female mortality is discernible as perhaps indicating that some further explanation is required. As regards this, Dr. Thorne remarks that 'the excess of diphtheria death which attaches to females over males from three to fifteen years of age, increases precisely as the age advances which fits them more and more to take some share in the care of home, and of relations during periods of sickness.' [2] And he further remarks that 'something may depend upon the full significance of the term "domesticity," and upon its taking account of those acts of affection and tenderness which, in their relation to the sick, characterise females during the period of girlhood, as well as in mature womanhood.' Both these considerations are deserving of considerable weight, especially as regards a disease such as diphtheria, in the dissemination of which close contact is known to play a conspicuous part; but it still appears to remain doubtful whether increased exposure to infection can be regarded as entirely accounting for the excess of female mortality, especially in the very early years of life.

[1] *Practitioner*, vols. xxxi. and xxxii.
[2] *Milroy Lectures*, 1891, p. 35.

Influence of Age.—The mortality from diphtheria is far greater in the age-group 0–5 years than at any other age-period. For the years 0–5 taken separately, it is decidedly lowest in the first year. Between the second, third, and fourth years there appears to be but little difference, though it is probably at its maximum in the fourth year. After that it begins to decline. During the next five years, however, i.e. the age-group 5–10, the mortality is still about double that at all ages, but it thenceforward falls considerably, and after the fifteenth year is comparatively insignificant. As regards liability *to attack*, Mr. Power has shown in his reports to the Local Government Board upon diphtheria outbreaks at Brailes (1876), at Radwinter (1877), and at Pirbright (1883), that the attack incidence of the disease, even apart from the influence of school attendance, is greatest upon children between the ages of three and twelve years. A similar result is also brought out by Dr. Thorne in his report upon diphtheria at Coggeshall in 1877. These reports are of especial interest with respect to the age-incidence of diphtheria, and also with respect to the influence of school attendance upon the dissemination of that disease, a subject which will be referred to later.

Cause.—The dependence of diphtheria upon an organism seems now to have been completely established, but for the evidence upon this point reference must be made to Dr. Klein's article. Certain factors which may be regarded as having indirect bearing upon the causation and spread of diphtheria must, however, be referred to here.

Looking at the geographical distribution of diphtheria in England at the present day, and remembering also that at different times outbreaks of this disease have occurred in localities exhibiting almost every conceivable geological variety, one would naturally conclude that geological circumstance had no influence upon diphtheria prevalence. And in the main, perhaps, this is true, as regards the more conspicuous geological features. But there is, nevertheless, a growing opinion among those who have had practical experience of diphtheria outbreaks, especially in rural districts, that a general dampness of soil, whether due to the quality of the soil itself or to its topographical arrangement, does favour diphtheria prevalence; though its influence in this respect may, of course, be masked by other dominant factors. Dr. Charles Kelly, among others, has drawn special attention to this.

Besides an influence due to general dampness of soil, there is a considerable accumulation of evidence in support of the view that special and continuous dampness of dwelling is similarly concerned with diphtheria. This appears to have been first insisted upon by Dr. Thursfield (*Lancet*, 1878), and has since been supported by other observers.[1]

As regards the actual dissemination of diphtheria, direct infection from case to case plays an important part. In this process the virus is, doubtless, often given off in the breath from the throat of the sufferer, and inhaled by the recipient; but actual contact, as in kissing, and the conveyance of the virus upon drinking-vessels and spoons, is no doubt responsible for many cases of infection. In addition to the throat and air-passages of the recipient, the poison may attack other mucous surfaces, as the conjunctiva, and wounds of the skin.

In connection with the spread of diphtheria by infection, the influence of school attendance is a matter of great importance. 'That some such influence obtained was one of the earliest observations following an inquiry into the etiology of the disease in this country,' writes Dr. Thorne in his 'Milroy Lectures;' and he then proceeds to discuss fully the nature and extent of this influence. For the detailed evidence upon this subject the reader must

[1] See also Dr. Copeman's article on 'The Influence of Soil on Health,' vol. i. p. 338.

be referred to the lectures in question, but the conclusions at which Dr. Thorne arrives are summed up as follows :—'School influence would appear, then, to be operative for mischief in a number of ways :—

'1. It brings together those members of the community who are, by reason of age, most susceptible to diphtheria. 2. The children thus brought together are placed, and remain for many hours of the day, in exceptionally close relation with each other. In this connection, it must be remembered that we are dealing with a disease, the communication of which from person to person is largely dependent on the closeness of the mouth and nares of the recipient individual to the faucial mucous surface of the individual by whom the infection is imparted. And when it is remembered that children are so placed in elementary schools as necessarily to be for long periods inhaling the lung exhalations of their fellows, and that collective singing and a kindred form of general intoning often form part of the school programme, it will not be difficult to understand how especially favourable such circumstances are to the transmission of a specific infection which has a primary seat upon the mucous membrane and in the secretions of diseased fauces. 3. The closer the aggregation, and the greater the lack of ventilation, with corresponding hindrance to the free movement of air, the greater is the risk. 4. Such faulty sanitary circumstances of the school-house and its surroundings, and such other conditions as tend to a condition of general ill-health, in that they induce sore-throat, favour the reception, by children so suffering, of any imported diphtheria infection. 5. There are ample grounds for believing that the aggregation of children in elementary schools constitutes one of the conditions under which a form of disease of particular potency for spread and for death may be, so to speak, manufactured. 6. The danger of school aggregation is by no means limited to the period in which the throat disease is acute. "Recrudescence" of the throat mischief in the individual probably tends to be favoured by conditions intimately associated with school attendance. And similarly, even in the stage of the disease after paralytic neuroses have supervened, infection may be communicated. . . . 7. The practices of kissing and of transferring sweetmeats from mouth to mouth—practices which are more common amongst girls than with boys—as also the joint use of drinking-cups, &c., must, according to experience, be credited with assisting in the diffusion of diphtheria amongst schoolfellows.'

The conclusion expressed by Dr. Thorne in No. 5, above, with reference to the manufacture, under certain conditions of aggregation, of a form of disease of particular potency for spread and for death, opens up a consideration of great importance and interest with respect to the etiology of diphtheria.

It has again and again been observed that outbreaks of typical and fatal diphtheria have been preceded by a more or less widespread prevalence of throat illness of a minor sort, and that even when true diphtheria has appeared upon the scene, the cases of throat illness in different families in the affected locality have presented great variety of type and severity, some of these being true and unmistakable diphtheria, others less typical, and at all events unrecognised as diphtheria at the time of their occurrence, though followed by the neuroses characteristic of that disease ; and others, again, cases apparently of quite trivial throat ailment. Moreover, such outbreaks of diphtheria following upon minor throat illness have frequently occurred in particularly isolated localities, and under general conditions which seemed entirely to preclude the likelihood of their having resulted from the importation of specific infection from elsewhere ; the whole circumstances seeming strongly to point to the view of the more severe malady having 'grown' out of the latter. And so long ago as 1878 Dr. Thorne, referring to such ex-

periences, remarked that they appeared 'to indicate the possible occurrence of what may perhaps be looked upon as *the progressive development of the property of infectiveness*.'

It is, no doubt, true that recently acquired knowledge of the etiology of diphtheria may be regarded as indicating the *possibility* of the specific microbe having been conveyed to isolated localities in ways not hitherto suspected, as, for instance, by domestic animals, or even birds. And it has also to be remembered that there is reason for believing that the poison of diphtheria may remain dormant in the soil or elsewhere for long periods of time, to be again called into activity upon the occurrence of suitable conditions of environment; so that some of the outbreaks of diphtheria in isolated localities, though not traceable to recent throat illness there or elsewhere, may still be connected with past diphtheria in the neighbourhood in question. But, at the same time, the frequency with which true diphtheria is observed to follow upon a prevalence of minor throat ailment is so great as to render it difficult to avoid the conclusion that the former is at times connected with the latter by an evolutionary process. And it has to be borne in mind, moreover, that even in the case of diphtheria occurrences dependent upon the return to activity of 'specific' micro-organisms left in the locality by some previous outbreak of diphtheria, as suggested above, such micro-organisms may, by a more or less prolonged dormancy or saprophytic existence, have largely parted with their virulence; and among the conditions of environment necessary for full establishment of pathogenic function, passage through a succession of especially suitable hosts may hold an important place.

In connection with the sudden appearance of diphtheria in isolated localities, the question of the aërial conveyance of the virus naturally arises. Upon this point the evidence is conflicting. It must be admitted that many cases occur in rural districts which seem, at first at least, strongly to suggest the conveyance of the poison through the atmosphere for considerable distances. On the other hand, the general behaviour of diphtheria in the dwelling and the hospital would seem opposed to this view, for although the infection is no doubt capable of being conveyed through the atmosphere, of the room for instance, for certain distances, yet close association and even actual contact with the sick seem to play a far larger proportionate share in the spread of diphtheria than in that of many other infectious diseases. Moreover, although, as Dr. Thorne says, 'no limit can as yet be assigned to the conditions, as to distance or otherwise, under which the diphtheria contagion can travel through the atmosphere and yet retain its potency for harm,' it will be seen from what has been said above as to other possible modes of origin of diphtheria in isolated localities, that 'it is pretty certain that many occurrences of diphtheria which some years ago might with plausibility have been held to be due to an aërially transmitted infection, would now admit of a different explanation.'

In addition to the methods of diphtheria dissemination already described, it is certain that the infection may adhere to clothing and the like, and thus be transmitted from place to place. It is now, also, well known that the diphtheria virus may be distributed by milk. This was first established by Mr. Power, in 1878, with respect to an outbreak of the disease in North London.[1] Since that time several other outbreaks in association with particular milk services have been brought to light, notably one at Hendon and one at York Town and Camberley, also reported upon by Mr. Power in 1883 and 1887 respectively. It is impossible here to enter into the details

[1] *Report to the Local Government Board on an Epidemic Prevalence of Diphtheria in North London*, 1878.

of these outbreaks, and the reports themselves should be studied. It is sufficient now to say that they have been the means of establishing beyond doubt the fact that milk may act as the vehicle for the dissemination of diphtheria infection. Mr. Power was also able to show good reasons for thinking that the quantity of the milk consumed in the different households, and the storage of such milk before consumption, were important factors in determining the incidence of the disease upon the various households receiving the infected supply. The quantity of milk consumed, he suggested, might operate either *as quantity* or by leading to the more frequent use of milk, and consequent repetition of the act of infection. The storing of the milk, he considered, might be harmful, as giving time for the multiplication of specific micro-organisms, and this Dr. Klein has since shown to be the case.

It is important, however, to note that in none of the instances above referred to could any means be ascertained by which it was likely that the suspected milk supplies had become infected by human diphtheria. This, as well as certain circumstances connected with the outbreaks referred to, raised the question as to whether the cows themselves, suffering, perhaps, from an ailment so slight in appearance as to pass unnoticed by those about the farm, might not have imparted infective quality to the milk. According to the recent investigations of Dr. Klein, it would seem that this question has now been answered in the affirmative.[1]

And this leads to the matter, already several times referred to, of diphtheria in the lower animals.

Of late years, evidence as to the participation of the lower animals in diphtheria has been accumulating. Various observers in studying outbreaks of human diphtheria have met with cases of concurrent throat illness of a like kind among domestic animals and birds, particularly cats, sheep, horses, fowls, turkeys, and pigeons; and in some instances strong evidence has been forthcoming that the animal disease in question was derived from or communicated to the human subject.[2] To the experimental proof of the occurrence of diphtheria in cows reference has already been made, and it may now be added that Dr. Klein has also established, by laboratory experiment, the fact of the occurrence of diphtheria in cats.

Drainage defects, filth accumulations, and the like, have very generally been held to have concern in the origin and propagation of diphtheria. For years, indeed, it was commonly believed that circumstances of this kind constituted the main cause of diphtheria, which was accordingly regarded as essentially a filth disease. It must be admitted that a view so widely held, not only by the public, but also by the medical profession, must, in all probability, have had some basis in fact. Nevertheless, the knowledge gained more recently as to the natural history of diphtheria has largely tended to modify the belief in question.

It may be conceded at once, as being in a high degree likely, that such unwholesome conditions do, by lowering vitality, both predispose to infection and add to the severity of the disease, if contracted. Moreover, it would seem reasonable to suppose that drain air, or, for that matter, coal gas, would, by giving rise to a relaxed and unhealthy condition of the mucous lining of the throat, and by thus inflicting injury at the special seat of invasion of diphtheria, increase the liability to attack by that disease in the event of exposure to specific infection. And, further, the possibility of the diphtheria

[1] See page 161, *ante*.

[2] See Report by Dr. George Turner in the *Report of the Medical Officer to the Local Government Board*, 1886. Also Report by Dr. Bruce Low on an Outbreak of Diphtheria at Enfield in the *Report of the Medical Officer to the Local Government Board*, 1888.

bacillus finding its way into sewers, and hence into houses imperfectly disconnected from such sewers, cannot be denied. But, on the other hand, it has to be remembered that modern inquiry has tended to emphasize the share taken by more direct infection in the dissemination of diphtheria—either infection from recognisable forms of the disease, or from those minor, and perhaps unrecognisable, forms which it is now known may be capable of giving rise to severe and fatal diphtheria. And other means of infection, which were hitherto unknown, such as by specifically contaminated milk, or domestic animals, have also to be borne in mind. It is therefore certain that, as in the case of supposed infection through the medium of wind currents, so here, many cases of diphtheria which it has been customary to attribute to drain defects—and drain defects, moreover, which notwithstanding modern improvements may probably still be detected in very many houses as well where diphtheria is absent as where it is present—might now, with due care, be traced to one or other of the causes indicated. Lastly, if we take a broad view of the matter, we see two strong grounds for discrediting the popular notion that drainage defects and other filth nuisances play a prominent part in the causation of diphtheria: 1. Diphtheria is steadily increasing, in spite of the considerable and progressive diminution in the unwholesome conditions of the sort in question, whereas the reverse is the case with regard to enteric fever, a disease which is known to be controllable by sanitary improvements. 2. The geographical distribution of diphtheria in England lends no support to the view that that disease especially affects those areas in which filth nuisances abound or defective sewers are the rule. In referring to the results of his studies of the geographical distribution of diphtheria, Dr. Longstaff says: 'The practical deduction suggested by these facts is, that the cause, or causes, of diphtheria should not be sought for primarily in any high development of civilisation, such as sewers, but rather in some condition associated with a more primitive mode of life. Again, privies and ashpits can hardly be important agents in breeding or disseminating the disease, or we should expect to find diphtheria exceptionally prevalent in those Northern towns where such nuisances reach their worst, whereas the contrary is the case.'

It has been suggested that diphtheria may be disseminated by the agency of drinking-water, but the evidence at present is against such being the case.

Assuming that the diphtheria bacillus may at times find a habitat in the surface soil, a rise in the subsoil water may, by expelling the ground air, lead also to the expulsion of the bacillus, and consequently have concern in the origination of some diphtheria outbreaks, as suggested by Mr. M. A. Adams; and this is a matter well worthy of future study.

Relation of Diphtheria to other Diseases.—Diphtheria, or what is certainly, from a clinical point of view, diphtheria, and is followed by the characteristic paralysis, is not infrequently observed in concurrence with, but more frequently in sequence to, both measles and scarlet-fever, and less frequently perhaps, enteric fever. Some interesting cases of associated scarlet fever and diphtheria will be found recorded in a paper by Dr. H. F. Parsons in the Epidemiological Society 'Transactions' for 1883–84, and the meaning of their association is also there discussed. Speaking generally, it may be anticipated that diseases, such as scarlet fever and measles, which especially lead to more or less temporary damage to the mucous membrane of the throat would predispose to the reception of the diphtheria poison, and it seems likely that this is the true explanation of many of the instances of diphtheria following upon the diseases in question. At the same time, the association between

diphtheria and scarlatina, at all events, seems at times to be an extremely intimate one, and in individual cases the co-existence of the two viruses in the same body is at least a possibility. Indeed, there would seem no theoretical ground for altogether denying the possibility of such a co-existence of the viruses extending beyond the single case, and giving a hybrid character to other cases.

When diphtheria is prevalent, there is very commonly an increase in the number of deaths registered as membranous croup. This, however, is apparently a matter of nomenclature and erroneous diagnosis, the so-called croup being, no doubt, laryngeal diphtheria. Similarly, diphtheria deaths are often registered as mumps, laryngitis, and tonsillitis.

Period of Incubation.—Most observers regard the period of incubation of diphtheria as being a short one, usually two or three days, but as liable to vary from one to six days or so. Some authorities, on the other hand, give a much wider variation, as, for instance, from one day to a fortnight. As regards unusually long incubation periods, Dr. Thorne points out 'the possibility of one of those mild sore-throat attacks from which adults at times suffer during diphtheria epidemics, and this without known inconvenience,' having preceded in the individual the attack which attracted notice; the latter being, in fact, recrudescence of the disease in the individual in question. It is also not unlikely that the period of incubation varies with the quality, and perhaps with the dose, of the virus, as well as with the condition, especially as to the throat, of the recipient. In milk epidemics the period of incubation seems usually to be short, a circumstance most probably due to 'conditions, such as storage of the supply, [which] have favoured the multiplication of the bacilli and subsequent formation of chemical poison, as also the direct reception of the poison into the stomach. . . .'

Period of Infectiveness.—This is variously stated by different observers as from fourteen days to eight weeks. Much, of course, must depend upon the circumstances of the individual case; but it may well be suspected that considerable harm in the way of the spread of diphtheria is done by underestimating this period, and allowing diphtheria convalescents to mix with the healthy, and perhaps return to school while still in an infective condition. Moreover, in this connection the observations of Dr. Astley Gresswell, which seem to show that in certain individuals diphtheria may, so to speak, become chronic, and subject from time to time, especially upon exposure to cold and damp, to recrudescence, are of the utmost importance, and should be carefully studied.[1]

Protection.—It is usually said that diphtheria confers no immunity against subsequent attack. Certainly, second and third attacks in the same individual are not unfrequently met with, but how far the later attacks are due to a fresh infection, and how far to rejuvenescence of a dormant virus left by an earlier attack, as suspected in some cases by Dr. Gresswell, remains an open question.

[1] 'Diphtheria as a Chronic Malady in Particular Individuals, with Liability in them to Recrudescence' (*Trans. Epidem. Soc.*, N.S., vol. v., 1885–86).

CEREBRO-SPINAL FEVER

Synon.: *Epidemic Cerebro-Spinal Meningitis; Cerebro-Spinal Arachnitis; Cerebral Typhus; Malignant Purple Fever; Spotted Fever.* Fr. *Méningite Cérébro-spinale Épidémique*; Ger. *Epidemische Meningitis*; It. *Febbre Cerebro-spinale.*

History.—The more important history of this malady dates from the year 1837. Medical records, however, bear testimony to its prevalence in Switzerland, France, and particularly America, during the early years of the century. Some French writers, indeed, have considered that they could trace it in the literature of far earlier times; but upon this point the evidence, according to other authorities, notably Ziemssen and Hirsch, is of a doubtful nature. However that may be, the disease appeared in epidemic form in the South of France about the year 1837, being apparently at this time first observed among the troops at Bayonne, but almost simultaneously at Foix and Narbonne. During the next five years it extended over a large part of France, spreading, it would seem, from the centres mentioned. In its progress through France its incidence upon the military population was conspicuous, and it is interesting to note, as bearing upon the mode of its spread, that upon several occasions its appearance in particular localities seems clearly to have been due to importation by recently arrived troops. The following is a brief summary of this French epidemic:—

About the time of, or shortly after, its appearance at Bayonne, the disease prevailed up the banks of the river Adour, in the department of the Landes, and among the troops at Bordeaux and La Rochelle. In the following year it broke out at Rochefort among soldiers who had arrived there from the Landes, and in the next year (1839) it appeared at Versailles, again among the same soldiers, who had been moved there from Rochefort. In 1839 it also broke out in the garrison at Metz, and reappeared at Bordeaux; in 1840 it broke out at Strasburg, and a second time at Bayonne; and in 1841 in the garrison at Nancy—being carried in the meantime by detachments of troops to a number of adjacent places, such as Schlettstadt, Hagenau, Buxweiler, and Wasselonne (Hirsch). At Laval the disease had broken out in the garrison in the spring of 1840, and thence, in the following winter, it was seemingly carried to Le Mans, appearing about the same time at Blois, Tours, Poitiers, and other places around. In 1840–41 it was also present in the garrisons of Brest, Caen, and Cherbourg; and in 1842 there were a number of cases in Paris.

It has already been seen, however, that simultaneously, or almost simultaneously, with its appearance at Bayonne in 1837 the disease had also broken out at Foix and Narbonne. From this centre, apparently (1838) it spread to Hers, near Toulouse, and to the troops at Nîmes and Toulon. About a year later it appeared in the garrisons at Avignon, Perpignan, and Montbrison, and in the years 1841–42 at Marseilles and Lyons.

After a general abatement throughout France from 1842 to 1845, several more or less extensive epidemics again occurred in that country between 1846 and 1850. In its reappearance the malady seems first to have broken out in localities which had been affected by it during the previous epidemic of 1837–42, such, for instance, as the garrisons of Avignon, Lyons, Nîmes, Toulouse, and Metz.

During the period under consideration (1837–50) cerebro-spinal fever was somewhat widely prevalent in Southern Italy, where it seems first to have occurred in 1839–40, and to have continued to prevail until 1845. In 1840, also, it broke out in Algiers, probably in connection, as Hirsch remarks, with its prevalence in France, and the French troops at Douera were among those who were early attacked. In 1842 the disease became prevalent in the United States, where it was first observed in Tennessee and Alabama, subsequently becoming epidemic in a number of other States between 1842 and 1850.

During the year 1843 there was a malignant epidemic at Corfu, and a minor epidemic at Gibraltar in 1844. In Denmark the disease appeared in 1845. In the latter year, also, cases occurred in the Dublin and Belfast workhouses,[1] and in Liverpool. A few cases were also observed in Ireland in 1850. From 1850 to 1854 there seems little record of cerebro-spinal fever; but the latter year saw the beginning of another widespread epidemic diffusion of it, and the disease continued active in one country or another until about the year 1876. During this period the countries most severely affected were Sweden, Germany, Russia, Greece, Italy, and especially America. It was present also, but in a minor degree, in Great Britain, Denmark, Norway, France, Switzerland, Austria, Hungary, Turkey, and Portugal, Jerusalem, Persia, and Algiers. It was earliest observed in Sweden, where some cases occurred at Gothenburg in 1854; but it did not become truly epidemic in that country until the following year. It then slowly extended over the south of Sweden, dying down during the latter half of each year, to reappear early in the following year. Each year, too, it travelled farther northwards, until 1858, when it had reached the latitude of 63° N. Subsequently to this the disease was more or less present each year, but with gradually decreasing intensity, and chiefly in localities in which it had previously occurred, until 1865, when it became again more prevalent. But it finally died out in 1867.

Early in 1859 the disease had broken out in Norway at Opdal, in the province of Hedemarken, which adjoins Sweden. If, as there seem grounds for thinking, Sweden had previously to 1854 been free from cerebro-spinal fever, the probability of the malady having at this period found its way there from Denmark, where it had been prevalent a few years before, is obvious.

The country which especially suffered during the period under consideration was the United States of America. The disease broke out in 1856–57, both in North Carolina and the New York State, and between the latter year and 1874, according to Hirsch, 'scarcely a year passed without its being seen over a larger or smaller area, its diffusion from first to last covering the whole country.' In the Civil War, it was particularly prevalent among the troops, attacking also the negroes in the Confederate army.

In Germany, the disease was observed on a small scale in Silesia during the year 1863, and became more conspicuously prevalent in the winter of 1863–64, when it appeared at many different places in Posen, Brandenburg, East and West Prussia,[2] and Pomerania, remaining 'epidemic therein until the end of the following winter' (Hirsch).

In Hanover and Brunswick it was epidemic in the winter and spring of 1864–65. During the same period, but commencing somewhat later, it was also present in Bavaria, Hesse, Baden, and Würtemberg. By the end of 1866 the German epidemic had spent itself, and the disease became limited

[1] See paper in *Dublin Quarterly Journal* of 1846 by Dr. Robert Mayne.

[2] A report by Dr. Burdon Sanderson upon the epidemic at Dantzic in 1865, will be found in the Annual Report for that year by the Medical Officer to the Privy Council.

to quite small epidemics and sporadic cases, chiefly in the colder seasons of the year.

As regards Austria-Hungary, some cases occurred in Vienna in the spring of 1863—that is, it will be remembered, the year in which we have record of its appearance in Silesia. Other prevalences occurred at Gömor in Hungary, and Pola and Trieste in Austria, during the three following winters. In the same year it broke out in Russia and Greece, afterwards spreading widely in those countries. In Italy it did not, apparently, during this epidemic, appear until 1873. As regards Great Britain, a rather important outbreak occurred, notably among soldiers, in Dublin and other parts of Ireland, during the winter of 1866–67. Several interesting papers upon this epidemic were read before the Medical Society of the Irish College of Physicians, and a long discussion upon them took place.[1] The outbreak produced considerable sensation, and some observers were inclined, in view of the high mortality which attended it, and the purpuric rashes which occurred in some of the cases, to regard it as a reappearance of the 'Black Death'—a view, however, which was soon abandoned. About the same time some cases were observed near Lincoln ;[2] but the disease does not appear to have been conspicuously prevalent in England.

Subsequently to 1876, cerebro-spinal fever, judging from the scanty records of it, seems to have generally abated. Some minor epidemics have since been observed, as, for instance, that in Fiji in 1885,[3] in Dublin in 1885–86,[4] and in the basin of the Mediterranean in 1887–88.[5] In 1890, also, some cases, apparently of this disease, occurred in the Eastern Counties of England, especially at Oakley, in Suffolk. The Oakley cases unquestionably constituted a small epidemic of a malady having for its main characteristic cerebro-spinal symptoms—vertigo, headache, great drowsiness, marked retraction of the head, and in some cases opisthotonos and subsequent paralysis. In certain instances other symptoms also suggestive of true cerebro-spinal fever were observed, such as conjunctivitis, herpes of the mouth, and a rash 'like chicken-pox.' There was, too, as Dr. Low points out, 'a definite group of cases, with associated scattered cases in the neighbourhood, but with no apparent connection between them,' such as has been commonly observed in respect to cerebro-spinal fever. In two points, however, the Oakley outbreak differed from some of the more typical epidemics of cerebro-spinal fever as that disease has been seen in other countries, viz. in its low fatality and in the frequency with which multiple cases occurred in the same household. But neither of these differences could be held as establishing a distinction from true cerebro-spinal fever. It has several times been observed that well-marked epidemics of this latter disease have been accompanied by numbers of cases exhibiting somewhat similar symptoms to the typical malady, but of so mild a character as almost to escape attention. There would, therefore, seem no reason why a particular localised epidemic should not consist wholly of such relatively mild attacks. As regards the multiple cases, although it has been stated of certain important and well-marked epidemics of cerebro-spinal fever that multiple cases in households have been conspicuous by their absence, yet this has not always been the case.

[1] *Medical Press and Circular*, June 5, 12, and 19, 1867.

[2] *Lancet*, 1867, 'On an Epidemic of Cerebro-Spinal Meningitis,' by G. M. Lowe, M.D.

[3] See the account in the *Trans. Epidem. Soc.*, Lond., N.S., vol. vii., by Mr. B. G. Corney.

[4] *British Medical Journal*, June 26, 1886.

[5] Notes by Dr. Thorne in the *Report of the Medical Officer to the Local Government Board*, 1888.

On the other hand, having in view the prevalence of influenza in this country during the early part of the year 1890, the protean character of that malady, and the fact that in the first instance its main assault appears to be upon the cerebro-spinal system; and coupling with these facts the previous relative freedom of England from cerebro-spinal fever, it might no doubt be suggested that the low mortality and multiple cases in the Oakley outbreak were rather suggestive of a form of influenza in which the cerebro-spinal symptoms were more than usually prominent, than of cerebro-spinal fever. This hypothesis might, perhaps, be thought to be strengthened by the interesting account given later, by Dr. Bruce Low, of another series of cases of 'anomalous illnesses' in certain localities in Northamptonshire. In these latter cases pneumonia was the more prominent feature, and although decided meningeal symptoms were observed, they do not appear to have been so constant or so conspicuous as among the Oakley cases. As at Oakley, they were characterised by a low mortality, and multiple cases in households were common.

Nevertheless, it is to be noted that, as regards age incidence, the Oakley and Northamptonshire cases apparently differed from influenza, and although not in accord with some experiences of cerebro-spinal fever in this respect, the balance of evidence seems to point to that disease rather than to influenza.

Mortality.—Cerebro-spinal fever has at times given rise to considerable mortality. According to Hirsch, 4,577 deaths were officially assigned to this disease in Sweden during the periods 1854–60 and 1865–70, in which it has been seen the disease was prevalent in that country. In the epidemic about the Lower Vistula in 1865, at least 1,000 persons died of the disease in the Circle of Dantzic during the first few months of the year.

As regards this country, reference to the Registrar-General's Annual Reports shows that the deaths recorded annually in England and Wales as due to 'cerebro-spinal fever' during the fifteen years, 1876–1890, have ranged from 18 in 1888 to 58 in 1878. These sporadic cases, however, 'scattered broadcast over the country,' cannot, as the Medical Officer to the Local Government Board suggests in his Annual Report for the year 1888, with any confidence be affirmed as of the same nature as those seen in epidemic form elsewhere, though there is, 'in the fact of the occurrence of any such deaths in England, reason for watchfulness on the part of Health Authorities and their officers.' It may also quite well be, as Dr. Bruce Low suggests, that true cerebro-spinal fever is of more frequent occurrence than is generally supposed, owing to fatal cases being certified as 'fever,' tubercular meningitis, &c., and non-fatal cases being mistaken for some other ailment.

Fatality.—The fatality in well-marked epidemics has usually been very great. According to Boudin's statistics, quoted by Sir John Simon, 809 deaths occurred among 1,304 patients—a fatality of 62 per cent. In the worst epidemics the fatality, Sir John Simon points out, 'seems to have been as high as 80 per 100.' Dr. T. W. Grimshaw, in Quain's 'Dictionary of Medicine,' gives the fatality in some of the American epidemics as 75 per cent., and states that among the Irish Constabulary it reached 80 per cent.

It is, however, not improbable that the estimates referred to are somewhat too high, for, as Sir John Simon points out elsewhere in the same memorandum, while cases of the gravest kind 'are occurring, perhaps, not very numerously in a place, sometimes within the same area, or scattered over a wider one, a large number of other persons will suffer slight indications of similar nervous derangement. Headache, vertigo, muscular discomfort in

the head and limbs, and attacks of chilliness, are the chief of these minor indications, which apparently bear to the graver cases the same sort of relation as epidemic diarrhœa bears to concurrent epidemic cholera.'

If these milder cases could have been estimated, the fatality would probably turn out to be lower than it has usually been stated to be.

Influence of Race, Sex, and Age.—So far as present experience goes, all races would appear to be subject to this disease if brought within the range of infection. In various American epidemics the negroes have suffered excessively, but whether this was due to special susceptibility *quâ* race, or to conditions of life, is doubtful.

It is usually stated that the disease is more frequent among males than females; Hirsch, however, is of opinion 'that there are no considerable differences discoverable' in this respect.

As regards age, cerebro-spinal fever has been known to occur in persons of all ages, though least frequently among those beyond middle life. According to Dr. Grimshaw, 'the disease usually attacks those approaching the age of puberty or in early adult life,' and 'robust males between the ages of fifteen and thirty are its favourite victims.' This latter statement, no doubt, seems to derive some support from the experience of military epidemics; but the incidence of the malady upon troops is clearly due to some factor other than age; and where the civil population has been attacked, children have often suffered severely. Hirsch gives a long list of epidemics in which children up to the age of about fifteen years have suffered almost exclusively, and he remarks that 'it has been much less common for patients of from twenty to thirty years of age to outnumber the children and youths.'

Influence of Climate and Season.—Hitherto cerebro-spinal fever has in the main been limited to temperate and sub-tropical latitudes, and there seems little evidence of its having prevailed extensively in the tropics, though, as we have already seen, it has occurred in the Fiji Islands. In all countries in which it has at present been observed in epidemic form it seems to have been decidedly most prevalent in the winter and spring, again and again dying down at the approach of warmer weather, to reappear with the ensuing cold season. In the case of Fiji, also, we learn from Mr. Corney that 'the period at which the epidemic prevailed was the cool season of the year, in 1885, an unusually cool one for Fiji . . . .'

Cause and Mode of Dissemination.—One of the most noticeable features in the behaviour of this disease, as judged by its past history, is its marked incidence upon troops and persons living together in public institutions, such as workhouses and prisons. The French epidemic of 1837 and the following years was largely among the military, and in America, Ireland, and other countries, the soldiers have at different times been observed to suffer heavily. Similarly, in certain epidemics it has been the inhabitants of prisons and workhouses that have almost exclusively been attacked.

This circumstance might at first sight be regarded as indicating infection as at least a prominent factor in the spread of the disease. Nevertheless, the detailed evidence, though somewhat conflicting, is generally held to be opposed to this view. Thus, with respect to the Dantzic epidemic, Prof. Burdon Sanderson reports that he met with no facts 'which afforded ground for believing that epidemic meningitis was capable of being communicated by personal intercourse,' while, on the other hand, he found evidence to the contrary effect. In the town of Dantzic, for instance, the average number of persons living in a house was, he states, eighteen, but he did not hear of a single instance, up to the time of his inquiry, of two persons suffering in the same house. Influenced by experience of the same kind, and by the

allied observation that those in attendance upon persons suffering from this disease, and other patients associated with such persons in general hospitals, are but very rarely attacked, most authorities have expressed themselves decidedly against the spread of the disease by infection. In this connection, however, the following remarks by the Medical Officer to the Local Government Board, in his Annual Report for the year 1888, are of much interest: 'The disease that was epidemic in Cyprus [1888] did not appear to be infectious from person to person, and in this respect it corresponded with the outbreaks that were observed in 1864–65 in villages on the Lower Vistula, and that were the subject of report by Dr. Sanderson to the Medical Department of the Privy Council (Eighth Report of Medical Officer). But for some years there have been observed in various countries (Landesbezirke) of Prussia (notably in certain towns of Oppeln County, in Upper Silesia), outbreaks of cerebro-spinal meningitis in which German medical observers believe they have detected a certain infectious quality, and in respect of which the Prussian sanitary authorities enjoin that every precaution be taken that is proper to be taken in the case of definitely contagious maladies.' The evidence at present, therefore, would seem to suggest, either that cerebro-spinal fever varies considerably as regards infectiveness, or that different maladies have so far been confused under one common name.

On the whole, as regards well-marked cerebro-spinal fever, the evidence perhaps rather goes to support the view expressed in 1865 by Sir John Simon, that 'if directly communicable from person to person [cerebro-spinal fever] is communicable only in a very low degree. Such communicability as is familiar to us with typhus, small-pox, and other eruptive fevers, cerebro-spinal fever does most assuredly not possess.'

According to Fagge, 'one suggestion is that a contagious principle is given off by the sick, but that it has to undergo some transformation or intermediate stage of its development, possibly in another animal, before it can infect a human being;' and he adds that it is stated, 'on the authority of Mr. Ferguson, Veterinary Surgeon to the Privy Council in Ireland, that on each occasion when the disease has prevailed in that country it has co-existed with an epizootic of the same nature among pigs and dogs.'[1]

But if not directly communicable, in any high degree at least, there are strong grounds for regarding the disease as communicable in the sense of being transportable from place to place by infected persons and, perhaps, infected things. By some the disease has been thought of as due to a widespread influence independent altogether of human movements, and in support of this view it has been pointed out that on particular occasions cerebro-spinal fever has appeared simultaneously at widely separated localities. Obviously it may be true, given the essential cause of the disease at the spots in question (left, perhaps, by some previous outbreak), that certain widespread meteorological conditions may determine a simultaneous outbreak in places remote from one another. Or, again, it may conceivably be that, under the circumstances of modern life, such widespread conditions are capable of imparting to micro-organisms not usually pathogenic. ability to produce cerebro-spinal fever. But it must be remembered that, as regards outbreaks of the kind in question, it is extremely difficult to exclude the possibility of importation of the disease by human agency, and there is nothing improbable in the notion of its simultaneous importation at different places.

On the other hand, a broad view of the history of the disease decidedly supports the view that human intercourse holds a very important place in its spread. Taken generally, the march of cerebro-spinal fever has not

[1] Fagge, *op. cit.*, i. 697.

been particularly rapid, and the disease has largely spread circumferentially from previously invaded districts. Lastly, as regards particular places, there has been very strong suggestion of importation.

Ziemssen, Hirsch, and some other authorities, are agreed that circumstances of life have concern in the prevalence of this disease, and that persons living under conditions of overcrowding, defective ventilation, and filth, are more liable to suffer than those more favourably situated. In many epidemics among the civil population the disease is said to have been confined, or almost confined, to the most insanitary parts of the towns invaded; and when troops have mainly suffered, the officers have often been observed to enjoy a marked immunity. On the other hand, Professor Burdon Sanderson did not associate the outbreak in the Lower Vistula with unwholesome conditions; and as regards the Oakley outbreak, Dr. Bruce Low found the conditions of life among those affected no better and no worse than those of their class generally.

Among other predisposing causes, experience of military outbreaks seems to show that excessive fatigue is of importance, and apparently it has sometimes been practicable to make an epidemic of this disease decline by lightening the men's duties.

Considerable importance has also been attributed by some observers to dampness of soil as an etiological factor in this disease, and it has been suggested, in fact, that cerebro-spinal fever is of malarial origin. Hirsch, on the other hand, opposes the malarial theory, pointing to the prevalence of this disease in high and dry localities. Conversely, he states as regards France, that 'it is precisely the great marshy districts that have been least touched by it.' As pointing to the same conclusion he also refers to the fact of cerebro-spinal fever usually occurring in the winter.

In view of the frequency with which cerebro-spinal fever has been observed to occur in the colder seasons of the year, cold has from time to time been regarded as its ultimate cause. The association between cold and this disease is, however, by no means so constant as to support such a thesis. There are not a few epidemics on record in which the disease has survived the cold of winter, continuing to spread, or in some cases breaking out, in the warm weather of late spring, or even summer (*see* Hirsch). Clearly, then, the relation between cold weather and cerebro-spinal fever, although no doubt in a sense real, is indirect. For the essential cause we must search deeper, and in the present day it will hardly be denied that the general features of the disease in question point strongly to a micro-organism as that cause. We are even perhaps justified in going so far as to suggest that the organism probably belongs to the class of 'facultative parasites,' being capable of thriving and multiplying outside human or animal bodies.

Suggestions of this are found in the fact that the disease has often broken out in districts which have suffered from previous epidemics; that upon its first introduction into a district it has on some occasions exhibited but a minor manifestation during the first season of its appearance, becoming decidedly more prevalent during the next cold season; that while it is not conspicuously infectious, it is apparently transportable; and lastly, that in not a few instances it has shown a decided tendency to cling to, and practically limit its manifestation to, the inhabitants of particular public institutions, such as barracks, and even to particular parts of these institutions. All these facts, though no doubt capable of other explanations, are at least consistent with the hypothesis of the ability of the micro-organism to carry on a saprophytic existence, perhaps in the soil, and perhaps also in the dust and dirt accumulated between the boards of barrack-room floors.

SIMPLE CONTINUED FEVER

Synon.: *Febricula.* Fr. *Fébricule*; Ger. *Febricula*; It. *Febbricola.*

From time to time cases of more or less transient fever are met with which are unattended by any definite and constant symptoms, other than those commonly associated with pyrexia. The difficulty of classing such cases with any of the more specialised fevers long ago gave rise to the notion of a 'simple continued fever'—a notion which apparently dates back to the days of Hippocrates, and has survived to our own time. 'But most physicians now believe that a disease without any morbid anatomy, without any known etiology, and without any definite or characteristic course or symptoms, cannot be admitted into a useful nosology.'[1] Moreover, it is now becoming generally recognised that the cases of feverishness grouped together under one or other of the above names are, in reality, due to a variety of causes. Even Murchison, who himself described a simple continued fever, remarks that 'many cases are designated simple fever, or febricula, which are in reality mild or abortive cases of typhus or enteric fever, or relapsing fever without a relapse, or catarrh with an unusual amount of febrile disturbance.' And such is no doubt the fact, the cases of so-called simple continued fever being largely made up of atypical examples of the particular fever prevailing at the given time and place.

Some cases, too, are doubtless symptomatic of gastric or intestinal disturbance, tubercular mischief, and other specific or non-specific causes.

In the following table, however, will be found the number of deaths recorded by the Registrar-General year by year from 1869 to 1891 as having, in England and Wales, been ascribed to 'simple and ill-defined fever.' Prior to 1869 the Registrar-General did not separate these deaths from those due to the other continued fevers.

TABLE X.—*Showing the Deaths registered in England and Wales during the Years* 1869–1890 *as due to Simple and Ill-defined Fever.*

Year	Deaths	Death-rate per million living	Year	Deaths	Death-rate per million living
1869	5,310	239	1881	1,159	44
1870	5,254	233	1882	1,016	39
1871	4,248	186	1883	963	36
1872	3,352	145	1884	768	28
1873	3,081	132	1885	662	24
1874	3,089	130	1886	605	22
1875	2,599	108	1887	507	18
1876	1,974	81	1888	436	15
1877	1,923	78	1889	413	15
1878	1,776	71	1890	361	13
1879	1,472	58	1891	325	11
1880	1,490	58			

On reference to the table it will be seen that the registered mortality from 'simple fever' has undergone a very marked diminution during the period in question—a diminution, in fact, very similar to that exhibited by the typhus mortality. This is, no doubt, partly owing to improved and

[1] Fagge, *op. cit.*, i. 207.

more careful diagnosis; nevertheless, it is probably also in no small degree due to the diminished prevalence of certain other fevers, such as enteric, and more especially typhus fever—diseases which, it cannot be doubted, were responsible for a considerable share of the deaths previously recorded as due to simple fever.

Murchison believed that the recorded 'simple fever' deaths were 'due, for the most part, to enteric fever with latent abdominal symptoms.' This, however, would hardly seem to have been the case.

Dr. G. B. Longstaff[1] has shown that the seasonal mortality curve of simple continued fever is quite unlike that of enteric fever. The curve of the latter disease has a well-marked autumnal maximum, whereas in the case of the simple continued fever curve 'there is not a trace of this.' On the other hand, Dr. Longstaff does find 'some sort of resemblance' between the seasonal curves of typhus and simple continued fever.

For the present the matter of main importance, from a public health point of view, is that mild cases of fever, which do not present any well-defined symptoms, should not be hastily classed as 'simple continued fever'—a separate disease being thus assumed, which, perhaps, has no existence. Careful inquiry will often throw light upon the causation of such attacks. If, for instance, one or other of the recognised fevers be prevalent in the neighbourhood at the time, and more especially if it should be ascertained that the 'febricula' patients have been exposed to likelihood of infection by such fever, there will be grounds for thinking it probable that the apparently simple feverish attacks are in reality but mild and irregular examples of the more serious malady. Under such circumstances, the interests of the public, as well as those of the patients themselves, obviously demand that the cases should be regarded as of the graver sort, all the precautions being observed which would be considered necessary with respect to the particular fever prevailing.

ENTERIC FEVER

Synon.: *Typhoid Fever; Pythogenic Fever; Gastric Fever; Low Fever; Infantile Remittent Fever.* Fr. *Fièvre Typhoïde, Dothiénentérie*; Ger. *Typhus Abdominalis*; It. *Tifo Enterico.*

History and Geographical Distribution.—There are few more interesting and instructive pages in the history of medicine than that which records the gradual differentiation of enteric from typhus fever—a differentiation only effected after prolonged and painstaking study of the pathology of these two diseases. 'The doctrine,' Hirsch remarks, 'of the so-called "abdominal typhus" counts among the acquisitions which medicine owes chiefly to the methodical investigation of morbid anatomy.'

It is often supposed that enteric fever is a disease of modern times. History, however, does not seem to support this view, but rather goes to show that it is the differentiation of the disease which alone is modern.

The late Dr. Murchison was of opinion that 'some of the descriptions of the Greek writers probably referred to enteric fever,' and he

[1] 'The Seasonal Prevalence of Continued Fever in London' (*Epidem. Soc. Trans.*, N.S vol. iv.). Reprinted in *Studies in Statistics.*

points out that in the course of two successive autumns Hippocrates 'met with many cases of fever of the continual type, characterised by diarrhœa, offensive watery stools, bilious vomiting, tympanitis, abdominal pain, "red rashes," epistaxis, sleeplessness, or a tendency to coma, delirium, and subsultus, irregular remissions, a lengthened duration, and great emaciation.' Whether or not such early writings are to be taken as referring to enteric fever, the descriptions of various observers in the seventeenth century, notably Spigelius, Panarolus and Baglivi for Italy, and Willis and Sydenham for England, seem to leave little doubt that the disease was prevalent in their day, several of them referring to intestinal lesions and a general course of symptoms highly suggestive of enteric fever. Both Willis and Sydenham, according to Murchison, regarded this fever as differing from *febris pestilens* [typhus]. In the eighteenth century there was an increasing recognition of a form of fever described variously as slow fever, nervous fever, or low continued fever, which differed from ordinary typhus in the insidiousness of its course, the attendant diarrhœa, and the tendency to prove suddenly fatal, and which was accompanied by inflammation or ulceration of the bowels. The more important accounts of this malady came from Hoffmann of Halle (1698–1728), Strother of London (1727–29), Gilchrist of Dumfries (1735), Huxham (1739), Sir Richard Mannigham, F.R.S. (1746), Riedel (1748), and Roderer and Wagler, who described an epidemic of morbus mucosus at Göttingen in 1760.

That a doctrine of enteric fever as a disease distinct from typhus was at this time gaining ground seems evident from the circumstance recorded by Murchison, that 'Dr. Erasmus Darwin, of Derby, in a letter addressed to Dr. Lettsom in 1787 proposes as a question for discussion at the Medical Society, "Whether the nervous fever of Huxham be the same as the petechial or jail fever," ' and that 'Dr. Willan, in 1799, observed that Cullen had "improperly comprised under the term typhus the slow or nervous fever described by Gilchrist and Huxham, which may rather be considered a species of hectic, and is not received by infection." '

During the early years of the present century an increasing amount of attention was bestowed upon the intestinal lesions met with in cases of fever. These lesions were particularly studied in France by Prost, Pettit, and Serres, the latter observers stating that the lesions were limited to the lower part of the ileum; Bretonneau, who showed that the disease was localised in the solitary and agminated glands of the ileum; and Louis, who first gave to the disease the name of *fièvre typhoïde*.

All these French observers, however, appear, according to Murchison, to have regarded the disease they were studying as really the same disease as the typhus of camps and armies. This was doubtless owing to the fact that during this period enteric fever was common in France, while true typhus had almost entirely disappeared. Hence it was not unnatural that, upon finding intestinal ulceration very generally present in cases which were then regarded as typhus, the French physicians should have been inclined to regard such lesions as associated with that disease.

During the same period, i.e. the first half of the present century, the foundation for the differentiation of enteric fever was being pushed forward in other countries. Hildebrand, in 1810, distinguished between so-called contagious typhus and non-contagious nervous fever; and other German physicians soon after followed on the same lines, among them Schönlein, who introduced for the latter disease the term *typhus abdominalis*, which is still adhered to in that country. In England, during the first twenty years

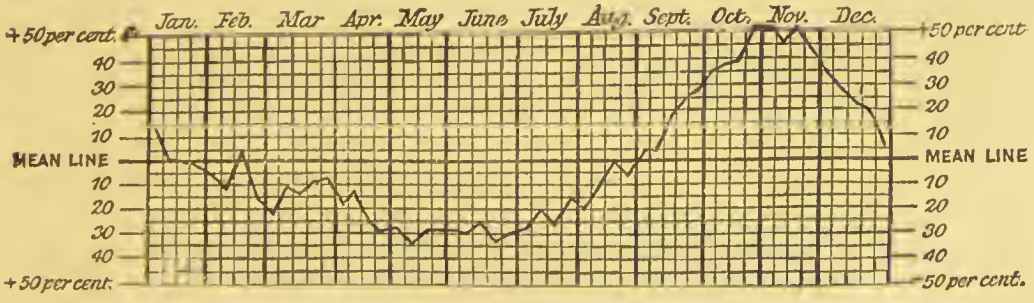
of the century several outbreaks of enteric fever seem to have been recorded, and to have been recognised as differing from typhus. The more important of these were an epidemic at Deal in the year 1806, recorded by Dr. Thomas Sutton; another at Paisley in 1811, recorded by Dr. Muir; and one at Newcastle in 1817, described by Mr. Henry Edmondstone. In 1826, Dr. Hewitt, of St. George's Hospital, published some important observations upon the pathology of this disease, which, Murchison says, met with unmerited neglect. They were 'published almost simultaneously with those of Bretonneau; and, like his, they showed that the seat of the lesion was in the solitary and agminated glands of the ileum.' Later on the separate nature of the two diseases was more fully recognised and insisted upon by Drs. Perry of Glasgow, Lombard of Geneva, Gerhard and Pennock of Philadelphia, Shattuck of Boston, U.S.A., and especially by Dr. A. P. Stewart of Glasgow. But it was reserved for Sir William Jenner, in 1849–51, to finally establish the non-identity of enteric and typhus fevers.

Sir William Jenner indicated more fully and more clearly than had hitherto been done the differences between the two diseases as regards symptoms and pathological lesions, and he also showed that the two diseases occurred independently of one another, were due to separate causes, and that the one did not communicate or protect against the other.

As regards geographical distribution, Hirsch describes enteric fever as an ubiquitous disease, and Murchison was evidently of the same opinion. It is certainly common throughout Europe, including Norway and Sweden, the Faröe and Shetland Islands, and Iceland. It is common also in North America, in Australia, and in India. It occurs in China, in Japan, and in various parts of Africa, notably Cape Town. On the whole, however, it has seemed hitherto to be less common in tropical than in temperate climates. Some allowance, too, has to be made for the probability that certain forms of malarial fever have been frequently mistaken for enteric fever. It seems certain, for instance, that remittent fever in the tropics frequently simulates enteric fever in a remarkable degree, though *post-mortem* examination shows the particular lesions of the latter disease to be absent. Many cases of the so-called typho-malarial fever are no doubt of this kind, as was pointed out in a paper read before the Epidemiological Society of London in 1886 by Dr. J. Edward Squire.

Influence of Season.—The influence of season, both upon the mortality and prevalence of enteric fever in London, is shown in the following curves, which are taken from the Registrar-General's Annual Summary for the year 1890.

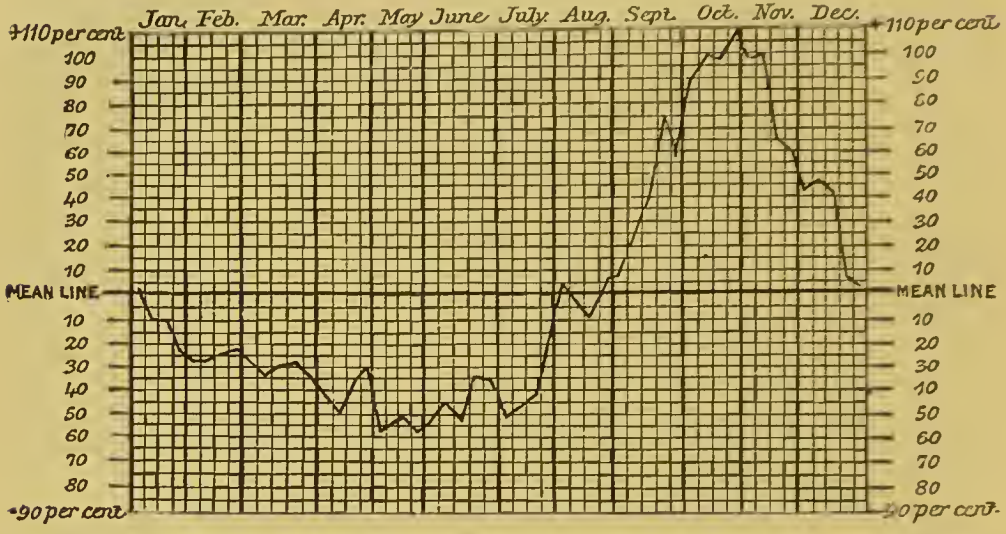
Enteric Fever.—London Deaths, twenty-two years, 1869–90.



For London, therefore, it appears that both the prevalence and mortality of this disease are greatest in the autumn, and least in the spring and early summer. A very similar relation to season obtains, according to Hirsch, for a number of European towns, including Paris and Berlin; and also for

Massachusetts and Boston. In New York, the maximum prevalence, Dr. Whitelegge says, occurs in September.

Mean Admissions of Enteric Fever Patients to the Metropolitan Asylum and London Fever Hospitals. (Sixteen years, 1875-90.)



Mortality.—The deaths from enteric fever have only been given separately by the Registrar-General since the year 1869. In the following table will be found the total enteric fever deaths recorded each year in England and Wales from that time to the present, and the corresponding death-rates per million of the population:—

TABLE XI.

England and Wales				England and Wales			
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1869	8,660	390		1881	5,529	212	
1870	8,731	388		1882	6,036	229	
1871	8,461	371		1883	6,078	228	216
1872	8,741	377		1884	6,380	236	
1873	8,793	376	374	1885	4,765	175	
1874	8,861	374		1886	5,061	184	
1875	8,913	371		1887	5,155	185	
1876	7,550	309		1888	4,848	172	179
1877	6,879	279		1889	5,011	176	
1878	7,652	306	277	1890	5,146	179	
1879	5,860	231		1891	4,875	168	
1880	6,710	261					

From this table it will be seen that, notwithstanding the considerable increase in population which has taken place during the period in question, the absolute number of deaths recorded in the country as due to enteric fever has latterly diminished considerably; while the mortality, relatively to the existing population, is only about half of that which occurred in the earlier years. And that this reduction in enteric fever mortality has been largely brought about by sanitary improvements there can be no doubt. In the Ninth Report of the Medical Officer to the Privy Council will be found the results of Dr. (now Sir George) Buchanan's well-known inquiry into the effect

upon the death-rate of twenty-five towns in England and Wales of the execution of various sanitary works, notably works in connection with improved drainage and water-supplies. In twenty-one of these towns Sir George Buchanan found an average reduction of over 45 per cent. in the enteric fever mortality subsequently to the execution of the works in question. In three instances there had been a rise in the enteric mortality, but in these cases it was clearly associated with conspicuous defects in the sewerage systems.

Case Mortality.—Of the 5,988 cases recorded by Murchison as admitted to the London Fever Hospital during the twenty-three years, 1848–70, 1,034, or 17·3 per cent., died, 'but, deducting those patients who were moribund on admission,' the fatality at that hospital during the years in question was 15·8 per cent., or about 1 in 6. Allowing, however, for the many slight cases which occur outside hospital practice, it may no doubt be safely concluded that the actual fatality is less than that indicated above.

Influence of Race.—Race *per se* seems to exercise no influence over liability to attack by this disease. Negroes, although apparently less liable to suffer in their native countries than non-acclimatised persons living there, become, Hirsch says, 'subject to the disease in America in no less a degree, *cæteris paribus*, than the white race.'

Influence of Sex.—As regards sex, it appears from the Registrar-General's Fifty-first Annual Report that, judging from the enteric fever deaths at all ages, taken together, the rate of mortality is slightly higher for males than for females. From the third year until the end of the twentieth, however, the female mortality is decidedly higher than the male. With respect to liability to attack at all ages, taken together, there appears to be little difference between the two sexes, though males are apparently rather more susceptible than females. 'Enteric fever,' says Murchison, 'attacks one sex as readily as the other.' But according to the Registrar-General, whose conclusion is based upon a study of the admissions to the London Fever and Metropolitan Asylums Board's Hospitals, it would seem that between five and twenty years of age more males are attacked than females. If this is usually the case, it follows that since more females than males die of enteric fever during the years in question, the fatality among females must be much greater from the fifth to the twentieth year than it is among males. The Registrar-General's conclusion, however, as to the greater liability to attack of males than females between the fifth and twentieth year is, as he points out, based only upon 5,716 cases.

Influence of Age.—Both liability to attack and to death by enteric fever are considerably modified by age. As to the former, it has been stated by some observers that susceptibility is greatest in the first years of life, and progressively diminishes thenceforward. It has to be remembered, however, that many alleged cases of enteric fever among infants are probably, as the Registrar-General has pointed out with regard to infantile enteric fever deaths, 'not due to that disease, but to some undetected cause, manifesting itself in feverish symptoms.' Murchison states that the disease is 'chiefly met with in youth and adolescence,' and that was certainly so with regard to his hospital cases. Of the 5,911 cases he records, the largest number, 26·86 per cent., were between the ages of fifteen and twenty, and 66·42 per cent. were between the ages of ten and twenty-five. After the age of thirty the cases became fewer and fewer.

The mortality in this country, that is, the deaths per million living as distinguished from the fatality, is, according to the Registrar-General, at its minimum in the first year of life for both sexes. It rises to the end of the

fifth year, and then falls, 'not inconsiderably for males, but quite insignificantly for females,' till the fifteenth year, after which it rises again, until its maximum is attained in the age period 20–25. Thenceforward it falls, becoming 'comparatively low through the remaining age periods.'

As in the case of typhus, so with enteric fever, the danger to life in the event of attack is greatest at the higher ages, that is, from fifty-five upwards; but, as Murchison points out, there is this important difference between the age fatality of the two diseases—that in the case of enteric fever it 'increases with age to a much less extent than in typhus, and the small rate of mortality (fatality) observed in early life in typhus does not occur in enteric fever.'

Period of Incubation.—The latent period of enteric fever is liable to considerable variation. Murchison and several other authorities give it as most commonly about a fortnight, considering at the same time, however, that it may range from a few days to twenty-eight or thirty. In the Guildford outbreak, reported upon to the Privy Council by Sir George Buchanan in 1867, it was eleven days. In the Caterham outbreak, reported upon by Dr. Thorne to the Local Government Board in 1879, it was fourteen days. A good instance of a shorter period is recorded in the report of the late Mr. Netten Radcliffe and Mr. W. H. Power upon the milk outbreak in the West of London in 1873. The patient, a child whose own family were not consumers of the infected milk, 'spent an afternoon (July 19) with a family supplied from this dairy, and during the visit drank nearly two pints of milk; on July 24 she was attacked by enteric fever.'

Protection.—The extent to which enteric fever confers immunity against subsequent attack is still doubtful. Murchison remarks that 'well-authenticated instances of persons contracting enteric fever a second time are more common than is generally believed,' and he refers to his own experience, and also to 'unequivocal' examples of second attack reported by Michel, Bartlett, Paul, and Dr. W. Budd. On the other hand, he points out that on questioning patients suffering from enteric fever 'it is rarely ascertained that they have had a previous attack;' and he refers to instances recorded by different observers, of second outbreaks in particular localities in which those attacked on the earlier occasion escaped during the later outbreak.

On the whole, there would seem little doubt that enteric fever does confer immunity, but for what length of time is a matter well worthy of further inquiry.

Cause and Dissemination.—The bacteriological evidence with respect to the causation of this disease will be found in Dr. Klein's article (page 168). It is sufficient here to say that the ability of the enteric fever virus to multiply in water and milk is a strong indication that it consists of a living organism. Further epidemiological facts support the view that this organism is decidedly 'facultative,' being capable of thriving and multiplying, not only in water and milk, but also in the soil.

Enteric fever is essentially a communicable disease, though the mode by which it is usually communicated differs widely from that of some other communicable diseases. It is now well established that in epidemics of enteric fever the virus is mainly given off from the body of the sufferer in association with the bowel excreta, and that it finds its way into the body of the recipient most readily by way of the alimentary system, as, for instance, along with water or milk. In this sense enteric fever is classable with cholera rather than with typhus, small-pox, and other so-called infectious diseases. Whether or not enteric fever is at all infectious in the popular sense, has been, and still remains, a subject for discussion. In the early days of the

recognition of enteric fever as a separate disease many observers, especially in France, held it to be highly infectious. Looked at in the light of modern experience of epidemics, however, we can perceive that the succession of a number of cases in the same house or locality, which to the observers in question seemed conclusive evidence of infection, may well have been often instances of multiple exposure at different times to one and the same cause. And nowadays, when the importation of a case of enteric fever into a village previously free from the disease is followed by a number of other cases among the inhabitants of the particular village, we have first of all to assure ourselves that infection of the later cases from the earlier has not been indirect —that is to say, due to contamination of water or milk supply by the bowel excreta of the first case, rather than to direct infection. Similarly, the cases to which the early advocates of the doctrine of infectiousness attached considerable importance—of nurses who contracted the disease while nursing patients in the patients' own homes—we should seek to explain, in the first instance, by exposure of the nurses to the same causes to which their patients owed their attacks. In this course we should be encouraged by the circumstance that the records of both fever and general hospitals seem to indicate that if the patients are treated away from the locality in which they contracted the disease, neither the nurses, nor other patients in the same ward, but suffering from different ailments, contract the disease, except upon very rare occasions.

Thus Murchison points out that 'during twenty-three years (1848–1870) 5,988 cases of enteric fever were admitted into the London Fever Hospital, but only seventeen residents in the hospital contracted the disease, and most of them had no personal communication with patients sick of enteric fever.' One of these was a laundress, who may have contracted the disease by handling the soiled linen of enteric fever patients; and 'twelve of the seventeen cases occurred subsequently to 1864, when various extensions of the hospital buildings led to a serious derangement of the drainage.' Mr. Shirley F. Murphy has since brought down the experiences of this hospital to 1878, with similar results. But Murchison also gives the following striking evidence. 'Since 1861 it has been the practice to classify the patients in the Fever Hospital in this way:—The typhus, relapsing, and scarlatina patients have been kept in distinct wards, whereas the patients suffering from enteric fever have been treated in the same wards with the many patients sent to the hospital who have not been the subject of any form of contagious fever. The two classes of patients have remained together, both during the acute stage of their maladies and in convalescence, in most instances for several weeks. The same night-chairs have been used for both classes, and the employment of disinfectant has been exceptional. The result has been this:—During nine years 3,555 cases of enteric fever have been treated along with 5,144 patients not suffering from any specific fever; not one of the latter has contracted enteric fever.' Dr. Cayley, in editing the third edition of Murchison's great work, remarks that 'the subsequent experience [down to 1882] of the London Fever Hospital is in complete accordance with these statements of Dr. Murchison.' It becomes clear, therefore, that if enteric fever is infectious at all, its infectiousness is, in ordinary circumstances, an insignificant factor in its dissemination. In the few recorded instances in which nurses in hospitals have apparently contracted the disease from patients—that is, the few instances in which no other explanation was forthcoming—it is possible that in dealing with the patients' excreta or the patients' soiled linen the specific poison has become attached to the hands of the nurses (or even become lodged beneath their finger-nails), and so has been conveyed

accidentally, as it were, to their mouths, and thence to their alimentary apparatus.

At the same time, it would not be safe to say that the disease is never conveyed by infection; and some great authorities, even during recent times, have held the view that such does occasionally happen. Sir William Jenner, for instance, has recorded two cases of enteric fever which occurred in medical students who, before the days of the registering thermometer, were engaged in frequently taking the temperature of enteric fever patients.[1] In the circumstances of the case these students must have constantly had their heads in close proximity to the patients, and in the absence of other explanation it would seem likely that to this fact their attacks were due. Even so, however, it does not follow that they acquired infection from the breath or skin exhalations of such patients. There would probably be some slight soiling of the patient's body or bedclothes with the specific bowel excreta, which when dry might become detached and distributed for a short distance upon the displacement of the bedclothes.

But although it is doubtless true that in epidemics the majority of *cases* of enteric fever are due to specifically contaminated water or milk—matters which will be dealt with shortly—we must not overlook other possible modes of the dissemination of this disease.

It has to be remembered that wholesale dissemination of enteric fever by means of contaminated milk or water supply, in that it gives rise to a large number of cases, tends perhaps to distract our attention from other, but no less real, distributers of the disease. There is no doubt, for instance, that the air of sewers and drains which has become specifically contaminated may, if allowed to find its way into dwellings through defective house connections, cause from time to time enteric fever among the inhabitants of such dwellings.[2] And this fact has, indeed, in many minds, given rise to the exaggerated idea that sewers, as such, have large concern in the production of enteric fever. That notion has, however, been discounted by Dr. Buchanan's inquiry, already referred to, and by the continued fall in the enteric fever death-rate which has since gone on side by side with the modern extension of sewerage works. It is to defects of the sewerage system, therefore, and not to the system itself, that mischief of this kind is due when it does occur. Nevertheless, defective sewers and house drainage are still, unfortunately, common enough, and must always be taken into consideration in endeavouring to trace the origin of obscure cases occurring in the same locality at irregular intervals of time.

Again, it is now generally admitted that the enteric fever organism is capable of thriving in the soil. It is a common experience in rural districts, and apart from sewerage systems, to observe the disease hang about a particular locality. Once introduced into a village, it will often recur regularly as the autumn comes round, invading households deriving their water supply from different wells, households not possessing community of insanitary conditions of a sort to explain the fever. This seems consistent with a specific organism capable of abiding in the soil, multiplying therein at its own proper season, and so contaminating the soil over considerable areas. In this way many different wells may become specifically polluted, and recurring outbreaks in villages have often been found to cease upon the provision of an entirely fresh water supply from some distant locality.

But if the microbe is capable of living in the soil, it may, as suggested by

[1] Quoted from Fagge, *op. cit.*

[2] See Reports on outbreaks of enteric fever at Croydon, Worthing, and York, by Drs. Buchanan, Thorne, and Airy.

Lindwurm (*see* page 336, vol. i. of this work), also find suitable conditions of existence in the dirt of rooms, as between the boards or stones of the flooring. To this cause may be due the tenacity with which the disease is well known to cling to certain barracks.

With regard to alleged relationship of variations in the level of the sub-soil water to enteric fever prevalence, the reader is referred to Dr. Copeman's article in the first volume of this work.

There are few facts more certainly established in the etiology of disease than the dissemination of enteric fever by specifically contaminated drinking-water. Quite early in the history of recognised enteric fever outbreaks of that disease were traced to polluted water by Drs. William Budd, Alfred Carpenter, and others.[1] And during more recent years a large number of further outbreaks have been proved to have depended upon the same cause. Space does not permit of even a brief summary of these outbreaks, and reference will therefore be made only to certain selected reports which illustrate particular points in the propagation of enteric fever by water.

In the Tenth Report (1867) of the Medical Officer to the Privy Council will be found some interesting reports of the kind. Thus, Sir George Buchanan describes there a severe outbreak of enteric fever at Guildford which was limited to the portion of the town over which a particular section of the public water-supply was distributed. Inquiry showed that about ten days before the outbreak occurred the portion of the town in question had been exceptionally supplied with water from a new well in the chalk, close to which well a leaking sewer was found to exist. In 1873 further progress was made in our knowledge of the circumstances under which enteric fever may be spread by water. Early in that year Dr. Blaxall was instructed by the Local Government Board to inquire into an outbreak at Sherborne, in Dorsetshire.[2] He found that the disease had been more or less present in the neighbourhood for some seven years previously, but that a notable increase had occurred in the number of new cases during the months of January and February, 1873; and, further, that during 'the first week in March there was a sudden and very great increase, and during the remainder of the month a gradual diminution.' Various possible causes for this increase having been inquired into and put aside, the process of exclusion led to the public water-supply as the probable source of the mischief. In connection with this water-supply it was ascertained that certain manipulations of the service had closely coincided with the variations in enteric fever prevalence. Thus, during the months of December 1872 and January 1873, the water 'was frequently shut off from the town at a point near the reservoir, and the same thing was done every night in February.' During the month of March this system of shutting off the water at night was discontinued. Thus the notable increase in the cases of fever in January and February coincided (allowing for the period of incubation) with the frequent intermissions in the water-service; the sudden and great increase in the cases in early March followed closely on the regular nightly intermissions of the water-supply; and lastly, the diminution in the number of attacks of fever was coincident with the discontinuance of the system of intermitting the water. Now, in Sherborne at that time this water was in some cases laid on direct to the closet pans by pipes supplied with taps. Some of these taps were broken. The effect, therefore, of turning off the water at the mains

[1] Parkes mentions the 'Schleimfieber' of Göttingen, in 1760, as having been attributed in part to impure water (Parkes's *Practical Hygiene*, p. 66).

[2] *Reports of the Medical Officer to the Privy Council and Local Government Board*, N.S., No. II. 1874.

would naturally be to establish a direct air communication between the water-mains (through the closet supply-pipes) and certain of the closet pans—in other words, as Dr. Blaxall remarks, 'the system of pipes for the water-supply became the means of ventilating the closet pans; if the trap happened to be broken or out of order, it became a means of ventilating the sewers; and if a pan happened to be full of excrement, that excrement would be sucked into the water-pipe.'

During the following year Sir George Buchanan[1] traced an outbreak of enteric fever at Caius College, Cambridge, to a broadly similar cause. The outbreak in question was mainly limited to a particular section of the college known as Tree Court. Tree Court derived its water-supply from the general 5-inch main of the Cambridge Water Company, which supplied the College, but by a special pipe from this main 'for Tree Court, and for no part but Tree Court.' Now it turned out that, unlike those in the rest of the College, the water-closets at Tree Court were supplied with water direct from the constant water-service, i.e. without the intervention of any disconnecting cisterns. And in addition to this, a small 'weeping-pipe' was given off from the closet supply-pipe to feed a miniature trapping-bend in the safe waste, which was taken direct into the closet trap. This arrangement is shown in fig. 1, taken from Sir George Buchanan's report. It is evident, then, that in the event of the water being intermitted, and the valve on the closet supply-pipe left open, there were here ample opportunities for admission to the water-pipes of foul air from the closet, or even liquid filth from the closet trap. It was further ascertained that about a fortnight before the enteric fever occurred in Tree Court the water-supply of this particular part of the College had, as a matter of fact, been temporarily cut off. It was also found on examination that the end of the weeping-pipe 'was crusted with a brownish matter,' and that internally it, and the supply-pipe between where the weeping-pipe came off and the valve, had also some little deposit. These deposits were submitted for analysis to Dr. Dupré, who came to the conclusion that 'water impregnated with fæcal matter must have entered the pipes.'

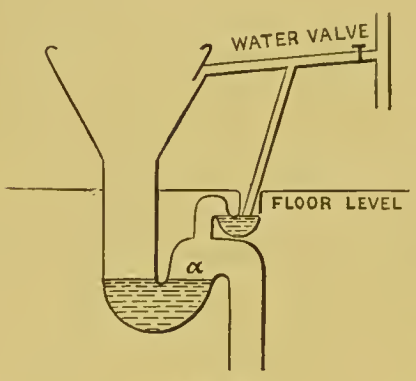


Fig. 1.

But a further link in the chain of evidence as to the origin of *specific* contamination of the water-supply of Tree Court was forthcoming. It turned out that between 'the unventilated sewer in Trinity Street' and the point marked *a* in the diagram there was 'not a single opening nor a single trap; nothing either to get rid of, or to attempt to keep back, the foul air' [from the sewer]. It is clear, therefore, that the water in both the large and small trapping-bends must more or less constantly have been charged with sewer air; and in the case of the smaller one 'a very small excess of atmospheric pressure in the down soil-pipe over the interior of the house would cause sewer air to bubble through the little bend; or some aspirating or syphon action . . . would empty this bend of its water.' Lastly, it was shown that shortly prior to the time of the outbreak in Caius College cases of enteric fever had occurred in houses draining into the Trinity Street sewer above the point at which it received the drainage of Tree Court, so that at the

[1] *Report of the Medical Officer to the Privy Council and Local Government Board*, N.S., No. II., 1874.

particular time in question the sewer air in the Tree Court soil-pipes must doubtless have been charged with the specific poison of enteric fever.

Clearly, therefore, there can be little doubt that when the water-supply of Tree Court was temporarily cut off (some one or more of the valves on the closet supply-pipes having probably been left open), specifically-tainted sewer air, and even similarly tainted water and filth from the trapping-bends had been sucked up into the Tree Court main pipe, and subsequently distributed with the water throughout the Court—'for fever attacked every staircase of the Court.'

The reports above quoted indicate very clearly some of the dangers of intermitting a water-supply. Before proceeding to consider certain other reports which in chronological order should follow, reference must be made to the admirable report by the late Mr. John Spear on an outbreak of enteric fever in the Mountain Ash U. S. D.[1] This outbreak was mainly limited to the area of distribution of a certain section of the public 'constant' water-supply. Careful inquiry enabled Mr. Spear to localise the probable spot at which contamination of the supply had taken place, and examination of the pipes at that spot showed that 'the main was carried, without any special precaution, immediately above, alongside, and even through old rubble drains, and when, in the course of the trenching, pipe-drains were cut through, no trouble appears to have been taken to replace them.' The main, which was leaky, was thus, 'at different points, from time to time bathed in refuse matters, and habitually, at certain points, in sewage-contaminated air.' Intermissions of the water-supply had taken place, and it is evident that, under the circumstances indicated, the pipes would 'take up foul air and liquid from these culverts and from the soil around.' But it is important to remember, as Mr. Spear points out, that such intermissions are 'not by any means essential to the introduction of foreign matters into water-pipes. Under various physical conditions, very powerful insuction of external matters into a full-flowing water-pipe can take place.'

Certain reports may now be quoted which illustrate the important fact that a degree of specific contamination of a water-supply so slight as to escape detection by the ordinary methods of chemical analysis may give rise to widespread outbreaks of enteric fever. The most important of these is the report of Dr. Thorne Thorne on an epidemic at Caterham. This report and its teachings have since been briefly summarised by Dr. Thorne himself as follows:—'In that case 352 cases of enteric fever took place at Caterham, Redhill, and certain intervening places, some 200 of the earlier cases being traced directly to the use, during a particular fortnight, of water derived from a deep well in which a man suffering from that disease had been employed, under circumstances which left no possible doubt that his excreta had got access to the well water. In this case it was estimated that, after all possibility of further pollution had come to an end, no less than 1,861,000 gallons of water had been pumped from the well during the fortnight in question; and Dr. Buchanan, in dealing, in his Annual Report for 1881, with the question of the amount of specific pollution which might suffice to render a potable water dangerous, showed that in this case the water could have contained no such amount as one grain of excremental matter per gallon, and he brought forward considerations tending to prove that an amount of specifically polluting matter so infinitesimal in quantity as to be altogether beyond detection by chemical analysis was fully potent for mischief.' In fact, as Dr. Buchanan tersely remarks, 'the chemist can, in brief, tell us of impurity and hazard, but not of purity and safety.' Other inquiries which

[1] *Report of the Medical Officer to the Local Government Board for the Year* 1887.

have resulted in supporting this conclusion have since been made. Among them may be mentioned a most interesting inquiry into an enteric fever epidemic at Houghton-le-Spring, which was reported upon by Dr. Page.[1] In this case the causal relation of the public water-supply to the outbreak was clearly shown, and the strongest evidence was forthcoming that the water in question was being polluted by filth which had travelled three-quarters of a mile through a fissure in the magnesian limestone. A sample of this water was at the time pronounced by an analyst to be 'a good water for drinking.' Comparatively recently, an outbreak of enteric fever in the Tees Valley has been clearly shown by Dr. Barry to have been distributed along with public water-supply pronounced by an expert analyst to be free from contamination. Clearly, then, although the analyst's evidence of pollution must always be gratefully received, his assurances of the purity of a suspected water must not be allowed to override other considerations tending to incriminate the water in question.

It has also to be remembered that enteric fever may be due to the use of contaminated water in the preparation of various 'drinks,' ices, &c. Thus, in 1882 Mr. G. H. Fosbroke traced an extensive outbreak of enteric fever to the consumption of lemonade, ices, and spirits which had been prepared or diluted with water from a sewage-polluted well.[2]

The probability of enteric fever being disseminated by the agency of milk was first pointed out by Dr. Michael Taylor in 1858.[3] In 1870 Dr. Ballard demonstrated the association of an extensive outbreak of enteric fever at Islington with the consumption of milk from a particular dairy, at which it was ascertained that a contaminated water was used for cleansing the dairy utensils. Three years later a widespread epidemic in Marylebone and other parts of the West-end of London was found by Mr. Netten Radcliffe and Mr. Power to be similarly distributed with a certain milk service; and here there was not only strong presumption that a contaminated water was used for dairy purposes, but that such water was actually contaminated by the excreta from a person suffering from enteric fever.[4]

Other outbreaks have since been traced to infected milk-supplies. In some of such instances the source of infection has been found in the use of contaminated water for the purpose of diluting the milk or cleansing the utensils, or in the presence of a person suffering from the disease at the particular dairy. But in an outbreak of enteric fever at Eagley and Bolton, traced, in 1876, beyond doubt to a particular milk service by Mr. W. H. Power, with the assistance of Mr. Robinson and Mr. Sergeant, the medical officers of health to the districts concerned, the question was raised as to the possibility of the milk obtaining infective quality from an ailment of the cow, as in the case of scarlet fever and diphtheria.

Since that date at least one outbreak has occurred in this country where, after minute inquiry, contamination of the implicated milk after it had left the cow appeared highly unlikely, and Dr. J. F. Allen, the Medical Officer of Health to Pietermaritzburg, has expressed his conviction, based upon his experience in Africa, that the disease may be caused in man by the milk of cows suffering from a similar malady.

[1] *Annual Report of the Medical Officer to the Local Government Board for the Year* 1889.

[2] See also a recent Report to the London County Council, by Dr. George Turner, on 'An Outbreak of Enteric Fever in the South-East of London.'

[3] *Edinburgh Medical Journal*, May 1858.

[4] *Report of the Medical Officer to the Privy Council and Local Government Board*, 1874.

Relation to Malaria.—The question whether or not enteric fever and malaria stand in any special relationship to one another has been extensively discussed. Boudin and others have maintained that a mutual antagonism exists between them, and that where malaria is common, there enteric fever is rare, or absent. This would seem to have been so in some localities; but it does not by any means universally hold good, and the more probable explanation of the phenomena as observed would seem to be that the rise of civilisation, by leading to the cultivation of the land and crude attempts at drainage, has tended to banish malaria, giving rise at the same time to the aggregation together of the people with—under the circumstances of the then defective sanitary arrangements—all its attendant pollution of air, water, and soil. To this, no doubt, was the increase of enteric fever due, and not to the disappearance of malaria. The question, moreover, is complicated by difficulty of diagnosis, and it is impossible to say to what extent the alleged disappearance of the one disease and appearance of the other may not in certain localities have been overstated. In some cases, during the earlier period of observation, enteric fever may, though present, have been mistaken for malaria; while, on the other hand, some of the later cases of supposed enteric fever may have been but an altered type of malaria.

De novo Origin of Enteric Fever.—In the foregoing pages it has been assumed that every case of enteric fever is derived from an antecedent case, human or animal, of like kind. The question as to the possibility of a *de novo* origin of enteric fever has, however, been keenly contested, and in a certain sense it must be regarded as still an open one.

The impartial inquirer can hardly fail to be struck by the weight of authority which has ranged itself on the side of the view that this disease may, and from time to time does, arise independently of an antecedent case. On the other hand, however, it has been maintained that the dependence of enteric fever on a specific poison implies that every case of that disease must be connected with an antecedent case of the same disease; that this must be as true of enteric fever as of small-pox; and that on this circumstance, in fact, depends its *specificity*. But this argument rests upon the assumption that there can be no degrees in *specificity*, no progressive development of specific quality, and this seems to lie at the root of the whole matter. It would be impossible, for instance, at present to allege that enteric fever never grows, as it were, out of simpler forms of diarrhœa.

The question must, indeed, be patiently worked out by the light of observation, neither the one theory nor the other being allowed to divert the attention of the inquirer from a faithful record of the actual facts. Every effort must, in all instances, be made to trace a causal connection between isolated cases of enteric fever and previous cases of that disease; while, on the other hand, failure after sincere endeavour to trace such a connection must be carefully noted, along with any observations suggesting other possible origin of the malady.

CHOLERA

Synon.: *Asiatic Cholera; Indian Cholera; Spasmodic Cholera; Malignant Cholera.* Fr. *Choléra Asiatique*; Ger. *Asiatische Cholera*; It. *Colera Asiatico.*

The connected history of epidemic cholera dates back only to the early years of the present century. Nevertheless it would appear, as Dr. J. M. Cunningham remarks, that cholera is 'no new enemy of mankind,' but has been known in India 'from the earliest times of which there is any record.'

Mr. Macnamara quotes a passage which is highly suggestive of cholera from the Sanscrit writer, Susruta, who lived before the Christian era, and he also considers that 'Hippocrates, Galen, and Whang-shooho are witnesses to the existence of cholera in their day, both in Europe and China; and their evidence is supported by a series of Grecian, Roman, and Arabian authors, bearing record to the fact of the presence of the disease in the various countries in which they lived.'[1]

The first notice of cholera in India by a European writer appears to be that of Gaspar Correa, a Portuguese, and refers to an extensive and fatal outbreak in the army of the sovereign of Calicut in the year 1503. Later in the sixteenth century outbreaks are mentioned by Portuguese and Dutch writers as having occurred at Goa. In the seventeenth century there is evidence of the presence of cholera at Batavia, Goa, and 'in London during the autumn of 1676, when Sydenham states that the cholera morbus was raging.'[2] Caution must, however, be observed in concluding that the early accounts of cholera on extra-Indian soil refer to true malignant Asiatic cholera.

Towards the close of the eighteenth century there were records, by Dr. Paisley and others, of epidemics of cholera in different parts of India. A limited number of cases also occurred in 1808–9, and scattered outbreaks from 1811 to 1814.

But the more important history of cholera dates from the year 1817. At that time commenced what proved to be the first recorded pandemic diffusion of the disease. Beginning in 1817 with a widespread prevalence in Bengal, cholera during the two following years extended throughout India and commenced to spread abroad. Travelling first (1819–20) in a southerly and easterly direction to Ceylon, Mauritius, Burma, China, Siam, Penang, Singapore, Sumatra, Borneo, and Java, it somewhat later spread in a north-westerly direction, through Arabia and Persia, and up the Caspian to Astrakhan (1823). It died out, however, as the cold weather came on.

In 1826 began a second and far more extensive pandemic. Starting from Bengal, the disease again spread (1829–30) to Russia by two routes, the one *viâ* Cabul, Balkh, Bokhara, and Khiva (caravan routes) to Orenburg; the other by Teheran and the shores of the Caspian Sea to Astrakhan, and thence up the Volga to Saratov and Kazan. In the meantime cholera was also spreading through Arabia, whence it advanced to Suez, Cairo, and up and down the Nile to Thebes and Alexandria respectively. From Russia it gradually extended over Europe and the greater part of America. It reached England in July 1831, having apparently been brought to the Medway by

[1] *A Treatise on Asiatic Cholera*, by C. Macnamara, p. 3.
[2] Macnamara, *op cit.*, p. 6.

ships from Riga. In October of the same year it broke out at Sunderland, as a result, it was believed, of importation by ships from Hamburg. It appeared at Newcastle and Gateshead in November, and at Haddington, in Scotland, in December.

In the following year, 1832, the disease was more extensively prevalent in Great Britain, many large towns in England, Ireland, and Scotland being invaded. To Canada the disease was apparently carried by Irish emigrants in June 1832, and rapidly spread throughout that country and also the United States. This pandemic did not entirely cease till the winter of 1837–38. It then died out 'at every point in the immense territory it had overrun in the period from 1826 to 1837. For the next ten years the soil of Europe, Africa, and America was completely free from it' (Hirsch).

The third great pandemic dates from the year 1846. Cholera, which assumed a wide epidemicity in India during the years 1840–41, extending also to Further India, the Philippines and China, appeared at Cabul in 1842. Thence, contrary to its custom, it extended from west to east through Peshawur, Lahore, and the North-West Provinces. In 1844 it reappeared at Cabul, spreading to Herat, Samarcand, and Bokhara in the same year. In 1846 it extended westwards to Asterabad and Teheran, and southwards to the shores of the Persian Gulf, whence it took a north-westerly direction along the valley of the Tigris, *viâ* Bagdad, to Diarbekir. In the same year the whole of the Arabian peninsula also became invaded, either from the Persian Gulf, or perhaps independently from Bombay *viâ* Aden. By 1847 cholera had spread from Persia to the shores of the Caspian Sea, whence it extended in a westerly direction along the coast of the Black Sea to Constantinople, and in a northerly direction to Astrakhan. At Orenburg, in the basin of the Ural, 'it joined hands with another invasion coming from Bokhara' (Hirsch). It now spread rapidly through European Russia, travelling up the Volga to Moscow and the shores of the Baltic. It also advanced from the Sea of Azov northwards up the river Don, and in a westerly direction to Odessa. During the winter the disease abated, but it reappeared over the whole of Russia and Poland in the spring of 1848. By the end of this year it had died out in Russia, but in the meantime it had invaded Germany, the Netherlands, Belgium, Great Britain, and America. As regards Great Britain, the first case heard of occurred in the person of a seaman named Harnold, who arrived in London on September 18 or 19 in a steamer from Hamburg, where cholera was prevalent prior to the departure of the vessel. This man died at Horselydown on September 22. The next case heard of was a man who slept in the same room with Harnold. During the first week in October some twenty-six cases, mostly fatal, occurred in London. Of these, eighteen were either on or close to the river Thames. About the same time (October 4, 1848) cholera appeared in Edinburgh. There the first cases were among some pilots who had been engaged in bringing in to Leith a ship from Cronstadt, where the disease was known to have been present in the summer. Also, in October cholera appeared in Hull and Sunderland, and again its appearance had been immediately preceded by the arrival of ships from Hamburg. The first known case in Ireland (December 2) was that of a man who had recently arrived in Belfast from Edinburgh, which city, as has been seen, was already invaded. To America the disease clearly seems to have been carried by German emigrants, who arrived almost simultaneously in two cholera-stricken ships, the 'New York' and the 'Swanton,' at Staten Island and New Orleans respectively.

During the winter of 1848–49 cholera apparently abated somewhat, but in the spring of 1849 it broke out again with increased vigour throughout the

greater part of Europe and America. In England the disease raged from April to December, 119 places being invaded. The mortality was greatest at Hull, where it reached the rate of 241 per 10,000 of the population. During this year France also was severely attacked. The disease appeared in Paris in March, and by the end of June 15,000 deaths had occurred. In 1850 Europe generally suffered little from cholera. The disease was, however, epidemic in Sweden, and also on the African seaboard of the Mediterranean, Egypt and Malta being heavily invaded. It was also widely diffused over America. During the next two years there was a marked remission in cholera prevalence in Europe, though scattered epidemics occurred from time to time, especially among the German Baltic ports. England remained entirely free during this period. But the following year (1853) saw an extensive resuscitation of the disease in Europe, and it became again widely diffused over a great part of the Continent. In the summer of this year it appeared in London, apparently as a result of importation from Germany, and later in the year it broke out at Newcastle, Shields, and Liverpool, also, it would seem, through importation from German ports. Later in the year it was epidemic in Manchester, Edinburgh, Glasgow, and Dundee. In 1854 it was very severely epidemic in Great Britain, and extensively prevalent over Europe generally, and America —in the latter case in consequence partly of fresh importations from Europe. For the following six years (1855–60) cholera continued from time to time more or less prevalent in Europe. Localised outbreaks occurred in England in 1855 and 1857. In 1859 cholera was imported into London, Hull, and Shields from Hamburg, but failed to spread. By the end of the year 1860, however, cholera had apparently died out in Europe. 'Regarded as a whole,' says Hirsch, 'this pandemic shows numerous fluctuations of intensity, the maxima falling in the years 1849–50 and 1853–55. In none of the intervening years was the disease altogether extinguished on extra-Indian soil, and there is no reason for attributing the fresh outbreak of 1853 in Europe, Africa, and America, to a new importation of the morbid poison from its native habitat. All the facts tell rather in favour of a continuous reproduction of the poison in extra-Indian countries, and that power of reproduction was exhausted only after the lapse of more than ten years.'

The fourth great pandemic began in the year 1863. As regards its invasion of Europe, this pandemic differed conspicuously from those already described, and 'is remarkable as marking a new epoch in the history of cholera, viz. its introduction by way of the Red Sea, wholly or in great part.'[1]

Starting from the basin of the Ganges, the disease, during the years 1863–65, became widely epidemic in Bengal, the North-West Provinces, Bombay, the southern parts of the Deccan, and Ceylon. It was also prevalent in China and Japan. In 1865 it was carried by ships to South Arabia (Hadramaut). It next broke out among the pilgrims assembled at Mecca (May 1865), by whom it was carried to many places, among them Suez (June). From Egypt the disease, during the next few weeks, spread to Malta, Marseilles, Ancona, Valencia, Constantinople, and other places. From these centres it extended through France, Italy, Spain, Turkey, Roumania, and Southern Russia. England was invaded in August of this year (1865), though only on a small scale, a few cases occurring at Southampton, and a localised outbreak (apparently connected with the Southampton cases) at Theydon Bois, in Essex. To Southampton the disease seems to have been brought from Alexandria. In the Western Hemisphere,

[1] 'Retrospect of the Successive Epidemics of Cholera in Europe and America' (E. F. Willoughby, M.D., D.P.H., *Epidem. Trans.*, N.S., vol. x. 1890–91).

the disease appeared in October 1865 at Guadeloupe shortly after the arrival of two ships from Marseilles and Bordeaux. Limited outbreaks also occurred in 1865 in Austria and Germany, and a more severe one in Belgium (Luxembourg).

In the following year (1866) the disease became extensively prevalent in Belgium, the Netherlands, Sweden, England, France, Italy, Turkey, and Russia. Scattered cases also appeared in Denmark, Norway, and Finland. It now, too, appeared in and became diffused over parts of North and South America. In England the disease was, during this year, imported at various places, notably Bristol and Liverpool, from Rotterdam; Southampton, by the P. and O. steamer 'Poonah,' coming from Alexandria *viâ* Malta and Gibraltar; Goole from Antwerp; and Shields from Hamburg. To North America cholera seems to have been in the first instance carried by Irish and German emigrants from Liverpool.

Although cholera in Europe and America now began to decline, some years elapsed before it disappeared. Thus, it was more or less present in Russia each year from 1867 until 1874, and in Germany in each of the same years, with the exception of 1869–70. In Switzerland it was present in 1867; in France in the same year, and also in 1873; in Austria in 1867, 1872, and 1873; Sweden in 1871 and 1873; South America in 1867–8, and North America (re-imported) in 1873.

The years of lowest epidemicity during this period were 1869–70, after which, up to 1873, the disease again became more prevalent. It is maintained by some that this renewed prevalence was a result of a fresh importation of the disease from India, and there would appear grounds for thinking that such was in part, at all events, the case. It seems certain that cholera became widely epidemic in North-West India after the great Hurdwar bathing fair in 1867, and that thence it spread through Persia into Russia in 1869 and 1870; so that the subsiding European epidemic would seem at least to have been reinforced by this fresh importation. Hirsch, however, takes the opposite view, and regards the renewed prevalence in Europe at this period as a resuscitation of the temporarily abated epidemic. By the end of 1874 cholera had disappeared from Europe, and did not return for a period of almost ten years.

In June 1884, however, cholera broke out at Toulon, possibly, it would seem, as a result of importation by French troops from Cochin China or Tonkin.[1] At the end of the month the disease had appeared in Marseilles,[2] and later in the year it spread over a large part of France. More than 880 deaths occurred in Toulon, and nearly 2,000 at Marseilles. As early as the end of June, cholera had appeared in Paris, but the serious prevalence of the disease in that capital did not take place until November, during which month there occurred there 938 cholera deaths. Altogether, during the year 1884 some 5,000 deaths from cholera occurred in France. In the meantime the disease had also appeared both in Italy and Spain, notwithstanding the extensive quarantine precautions which were taken to prevent its importation to those countries. In Italy, it seems first to have broken out at Spezia. Before the end of the year 858 Italian communes had been more or less invaded, and, according to the Official Report of the Minister of the Interior,

[1] For the detailed history of cholera in Europe during this and other recent years, see the Reports and Papers on Cholera submitted by the Medical Officer to the Local Government Board as a supplement to the Board's *Fifteenth Annual Report*; also the various Annual Reports of the Medical Officer to the Local Government Board.

[2] There appears some evidence that cholera was present at Marseilles in the year 1883. See footnote to p. 9 of the *Annual Report* for that year of the Medical Officer to the Local Government Board.

as many as 27,030 attacks and 14,299 deaths had resulted. The disease was most severe at Naples, in which city there were 7,086 deaths. It is notable that Rome, with its excellent water-supply, almost entirely escaped, only thirteen cases and six deaths being recorded for the whole province. In Spain the epidemic was not, during 1884, of a very extensive character. In all there were 274 cholera deaths in that country, 215 of which occurred in the province of Alicante.

During this year (1884) cases, apparently of cholera, were three times brought in ships to England, but no spread of the disease occurred. Germany, also, practically escaped.

By the late autumn or early winter the disease had died down in France, Italy, and Spain. But it reappeared in each of those countries in 1885. This time Spain was heavily attacked. The earliest recorded cases occurred in the province of Valencia during the month of February, but the disease did not become widely diffused until June. It then increased through the months of July and August, beginning to decline in September, but it had not died out at the end of the year. During the period February 5 to December 31, 1885, forty-six provinces had been invaded, the total recorded cases numbering 338,685, and the deaths, 119,620. In Italy the disease became epidemic in a number of provinces in August, and before the end of the year 3,459 deaths were recorded, 2,959 of which occurred at Palermo. As regards France, cholera was, during 1885, most fatal in Marseilles and Toulon, at which places 1,039 and 314 fatal cases occurred respectively, the main mortality occurring in the month of August. Later in the year, however, cholera appeared in the province of Finistère, and some hundreds of deaths occurred there, notably at Brest.

Several suspicious cases of diarrhœa were brought by different ships to England during 1885, but this country again remained free from epidemic cholera.

By the spring of 1886 cholera had practically disappeared from France and Spain. In Italy, however, it was again prevalent, especially on the Adriatic seaboard, and more than 6,000 cholera deaths were recorded. Austria-Hungary now also became somewhat severely invaded, especially at its Adriatic ports, the deaths numbering over 2,000. The disease still lingered in the kingdom of Italy in 1887, mainly in the island of Sicily; but by the end of that year the European epidemic which had begun in 1884 came to an end.

Europe now remained free from cholera until 1890, when the disease suddenly reappeared at Valencia. At this time cholera was steadily advancing through Persia and along the Euphrates Valley, but there seemed no sufficient grounds for connecting the Valencia outbreak with that circumstance, and it was suggested, on the other hand, that the reappearance of the disease in Spain was due to excavations in ground infected in the previous epidemic by cholera evacuations.

In the meantime (1890) cholera continued to advance in Persia, Turkey in Asia, Syria, and Arabia, reaching Tabrez, Diarbekir, Aleppo, and Mecca.

About June 1891 it again broke out at Aleppo and the surrounding villages, appearing later at Damascus, Antioch, and other places. In Mecca also it reappeared, causing upwards of 11,000 deaths. From Mecca it was carried by pilgrims to Medina and Jeddah.

Towards the end of 1891 cholera, which had apparently extended from the neighbourhood of Peshawur, broke out in Cabul. After a temporary abatement during the months of January and February 1892, it reappeared there in March. In the same month it was also severely prevalent at Herat,

and two months later at Meshed. Later still, by some two months or more, it reached Teheran. But in the meantime (the beginning of June) it had broken out in Askabad, whence it quickly spread along the route of the Trans-Caspian Railway and across the Caspian Sea to Baku. From Baku it spread westward to Tiflis. being carried northward, at the same time, by steamboat traffic up the Caspian Sea to Astrakhan, at the mouth of the Volga, where it appeared on June 30. By July 6 it had broken out at Saratov, 500 miles up the Volga, and a little later at Kostroma. By the first week in August a number of the Central Russian provinces were invaded. By August 16 the disease was established in St. Petersburg, and by the 23rd the invasion of Hamburg was officially announced. To Hamburg the disease was probably brought by Russian emigrants *en route* for England and America. Later on cholera appeared at Antwerp, Amsterdam, Rotterdam, and in Galicia, cases also occurring in Brussels, Berlin, and other places.

But in addition to the cholera above referred to, which undoubtedly came direct from the East, there was also throughout the summer (1892) a sustained and extensive prevalence of cholera in France. The disease appeared in the suburbs of Paris at the very commencement of April, the city itself becoming invaded a week or two later. Subsequently cases appeared at Havre, Rouen, and other places. Although this French cholera did not exhibit a tendency to very rapid spread, its fatality was considerable, and there are strong grounds for regarding it as true Asiatic cholera. Its early appearance, however, and the absence of evidence of its direct importation from the East, make it possible, as Mr. Macnamara suggests, that it was a revival of the European cholera of 1884–87.

The mortality from cholera in Europe during 1892 was very great. In European Russia to November 30, 132,700 deaths occurred. In Hamburg, according to official returns, the cases in the first nine weeks of the outbreak numbered 17,989, and the deaths, 8,261. As regards France, the deaths from April to September 14 in Paris and the suburbs, in the Department of the Seine, amounted to over 1,400.

The experience of England during the epidemic was decidedly encouraging. Two days after Hamburg had been declared infected, three cases of cholera from that city arrived at the port of London in the s.s. 'Gemma,' and by the middle of October some twenty-nine undoubted cases had been brought to this country. But in no instance, so far as is known, did the disease extend beyond the imported cases.

The points to note with regard to the cholera pandemic of 1892 are: (1) That the disease again travelled along main lines of communication, and, in many instances, in conspicuous association with the movements of persons along those lines. In this connection it is noteworthy that within a month 'of the recognition of cholera at a town on the Trans-Caspian Railway it (cholera) had penetrated to the heart of Russia in Europe, the transit from Central Asia having taken as many days as, before the creation of railways and steamboat lines, it took months.'[1] (2) That cholera has again exhibited its tendency to flourish where unwholesome conditions abound, and that as regards Hamburg, and Paris and its suburbs, at least, the victims have for the most part been persons consuming contaminated water. (3) That attempts to stay the march of the disease by measures of quarantine have again, in some instances, signally failed, while, on the other hand, the more practicable and reasonable system of medical inspection adopted in this country, although it does not claim to be an absolute protection against the

[1] 'The Route of Asiatic Cholera in 1892,' by Dawson Williams, M.D. (*British Medical Journal*, Sept. 17, 1892).

importation of the disease, has nevertheless been attended with success in that respect.

Before leaving the subject of the history of cholera, reference must be made to one or two circumstances brought out by that history. In the first place, the favourite, and possibly original, home of the disease would seem to be India, and especially Lower Bengal—a region from which cholera is practically never absent, and from which it has again and again extended in epidemic or pandemic diffusion over wide areas of the earth's surface. In such epidemic or pandemic excursions cholera has tended especially to advance along main lines of human communication, such as caravan routes, navigable rivers, and the lines of sea traffic. Its appearance in previously uninvaded localities has often been found to have been closely preceded by the arrival of persons from infected districts; and lastly, extensive diffusions of the disease have at different times followed upon the congregation and subsequent dispersion of pilgrims, as at the great Hurdwar Fair, in India, and at Meschid and Mecca. It will have been observed from the historical sketch above given that, in its marches from India to the West, cholera has followed certain well-defined routes, which have been described as the Central Asiatic, the North Persian, the Persian Gulf, and the Red Sea routes. The Central Asiatic route advances through Cabul, Balkh, Bokhara, and Khiva, to Orenburg in European Russia; the North Persian, *viâ* Cabul, Herat, Meschid, Astrabad, and Teheran, and thence up the Caspian Sea to Astrakhan, and through Tabrez to Erzeroum, Trebizond, and along the Black Sea; the Persian Gulf route passes up the shores of the Gulf and north-westwards, along the river Tigris to Baghdad and Diarbekir, and the Euphrates to Aleppo. The Red Sea route, first followed in 1865, passes from India by sea to South Arabia, and thence along the shores of the Red Sea to Suez, Cairo, and the Mediterranean.

As regards geographical distribution little need be said. At one or another time cholera has extended, as we have already seen, widely over the earth's surface. In its marches, however, it has, so far, spared certain localities. Thus, it has never yet invaded Australia, the East Coast of Africa south of Delagoa Bay, the islands of the Pacific Ocean, the islands of St. Helena and Ascension, Iceland, the Faröe Islands, the Hebrides, the Orkney Isles, Lapland, and some other places (Hirsch). Even in invaded continents certain places have enjoyed a marked degree of immunity. Exemptions of this sort are probably due, either to the relatively little communication between the places in question and the continent of India, or to the enjoyment by such places of special sanitary advantages, more particularly pure and wholesome water-supplies.

Mortality and Fatality.—The mortality from cholera is often enormous. As regards this country, the deaths resulting from the 1831–32 epidemic were estimated at over 30,000. They were not, however, accurately known, as registration had not then been introduced. In the next invasion of Great Britain (1848–49) the mortality was very great. Thus, in the year 1849, when the epidemic was at its height, the cholera deaths registered in England and Wales amounted to 53,293, which was equivalent to a rate of 3,033 per million of the population. There was also during this year a notable increase in the 'diarrhœa' mortality. It is, however, satisfactory to observe that upon each of the two later invasions of this country (1853–54 and 1865–66) there was a marked decline in the mortality. In the year 1854 the cholera deaths were 20,097, giving a rate of 1,080 per million against 3,033 of the year 1849; and in the year 1866 the deaths still further fell to 14,378, or 672 per million. Since the latter year England has remained free

from epidemic cholera. In each English invasion the prevalence and mortality of the disease have been far greater in the second than the first year of the epidemic.

The fatality of cholera is also very high, usually ranging from 30 to 50 per cent. of the attacks, and it may be even higher. In 1892 it seems to have been about 45 per cent. both for the whole of Russia and for Hamburg. The fatality of cholera is said to be greater at the beginning than during the later stages of an outbreak.

Influence of Climate and Season.—That Asiatic cholera is largely influenced by climate there seems no room for doubt, for although it is true that the disease has shown its ability to diffuse itself widely under various conditions as to climate, it has not shown an equal ability to permanently establish itself under such diverse climatic conditions. On the contrary, in temperate climates it tends to die out in the course of a few seasons. Moreover, that a certain degree of heat favours the activity of the poison is sufficiently evidenced by the fact that in Europe the disease has generally attained its greatest prevalence in the months from June to August, dying down during the winter, often only to reappear in the following summer. The fact of there having been certain exceptions to this order of events does not alter the bearing of the usual experience. 'As a general rule,' says Macnamara, 'it may be stated that cholera will not extend during the cold of a European winter, or even of our Punjab cold season.'

As regards rainfall, it would seem that a moderate degree of rain favours, and is probably essential to, cholera prevalence, though excessive wet arrests the spread of the disease. Further information upon this subject, however, will be found at page 343, vol. i. of this work.

Influence of Race.—There is a general consensus of opinion among authorities to the effect that the negro race is especially liable both to attack and death by cholera. This has been particularly noted on the East Coast of Africa, at Mauritius, Réunion, and other places. But as to the relative susceptibility of other races little seems to be certainly known.

Of Sex.—Judging by the experience of the three cholera epidemics which have occurred in England during registration times, the mortality at all ages taken together is rather greater among males than females, the male mortality being the higher up to the age of fifteen years and after that of sixty-five.

The fatality (or case mortality) is greater for females than males in the age period 0–5 and during the age periods 10–15 and 15–25; at all other age periods it is greater for males.

And of Age.—As regards age apart from sex, the actual number of deaths is much greater during the age period 0–5 than at any subsequent age period, and of the years included in the period 0–5, it is greatest in the second year. If the mortality at different age periods is considered in relation to the numbers living at those periods, it appears that it diminishes from 30·1 per 10,000 living to 12·9 and 7·0 respectively during the first three lustres of life (0–5, 5–10, and 10–15). It then remains almost stationary, though increasing slightly, for the next ten years, after which it increases steadily with each succeeding decennial period until it reaches 43·6 for the period 75–85.[1]

The case mortality is also greatest in children and old persons.

Cause and Modes of Dissemination.—The ultimate dependence of cholera upon microphytic life-processes is here provisionally assumed. The evidence with respect to the bacteriology of cholera is, however, dealt with

[1] See *Report on the Cholera Epidemic in England*, Dr. W. Farr (Supplement to the *Twenty-ninth Annual Report of the Registrar-General*).

at page 175, *ante*. Among the circumstances affecting the development and diffusion of cholera, conditions of locality are of great importance. On a large scale this is seen in the constant presence of the disease in Bengal, its so-called 'endemic centre.' On a smaller scale it is seen in the frequently observed fact that certain localities, even particular parts of towns, have been severely invaded in successive epidemics, while other more or less circumscribed localities have repeatedly escaped serious invasion, notwithstanding repeated importations of the poison.[1]

It is doubtless true as regards European experience, that in the instances in which particular localities have again and again been heavily attacked, a partial, and in some cases, probably, a complete explanation of the fact is to be found in the existence at such places of polluted water-supplies or other unwholesome conditions. But, in a general way, there is much evidence of the influence of other factors, such as elevation; nature and character of the soil, together with its natural purity or organic contamination; moisture and temperature of the soil, and variations in the level of the ground-water. These matters, however, are discussed in the first volume of this work (p. 339).

That sanitary defects are conducive to cholera prevalence and mortality is unquestionable. Such defects no doubt operate indirectly by inducing a lowered standard of health with diminished power of resistance to infection, but their main danger lies in the specific contamination of air, soil, food, and especially water, to which they give rise. Mr. Macnamara speaks of the general agreement of Anglo-Indian authors 'that cholera, when extending over a country, often settled on the inhabitants of low-lying, ill-drained, and overcrowded localities.'[2] Sir John Simon, commenting in his Ninth Annual Report to the Privy Council upon the experience of the cholera epidemic which England was then (1866) passing through, remarked: '. . . The diffusion of cholera among us depends entirely upon the numberless filthy facilities which are let exist, and specially in our larger towns, for the fouling of earth and air and water, and thus secondarily for the infection of man, with whatever contagium may be contained in the miscellaneous outflowings of the population. Excrement-sodden earth, excrement-reeking air, excrement-tainted water, these are for us the causes of cholera.'

But the conditions so far referred to are conditions tending to promote the activity and diffusion of the cholera organism when it is present in the locality. It has, however, already been seen that the conspicuous feature in the behaviour of cholera during the present century has been its liability at frequent intervals to escape from its endemic centre, and sweep over more or less wide areas of the earth's surface. Various theories of 'cholera waves' and the like have been advanced to explain these extensive marches of the disease, but, the more closely the matter is studied, the more evident does it appear that the actual migration of the disease is accomplished by human agency.

Not only does a survey of cholera pandemics exhibit their tendency to advance along the lines of human communication, but a detailed study of particular outbreaks sufficiently often discloses the fact of the importation of the disease by infected persons or their infected clothing.

Doubtless the variations in the amount of cholera prevalence in India from time to time, and the periodical diffusions of the disease from that country, indicate the operation in particular years at the endemic centre, and, perhaps more or less throughout the march of the disease, of meteoro-

[1] See Hirsch, *op. cit.*, vol. i. p. 439.

[2] Article on 'Cholera,' Quain's *Dictionary of Medicine*.

logical or other conditions which are favourable or the reverse to the multiplication of the cholera organism or to the promotion of its pathogenicity; but the evidence goes to show that the actual transport of the organism is effected by human agency.

The mode by which the disease is communicated, however, differs notably from that of small-pox, typhus, and other typical infectious diseases, and more closely resembles that of enteric fever. It is not, as with typhus, those who are in attendance upon the sick who are especially singled out for attack. Such persons do not, as a rule, appear to suffer in greater proportion than the other inhabitants of the infected district. On the other hand, there is abundant evidence that cholera may be communicated by drinking water contaminated with the bowel discharges of persons suffering from the disease. With respect to this evidence the reader must be referred to the article on 'Water' in the first volume of this work (p. 267).[1]

It is not, however, maintained that water-carriage constitutes, even in this country, the only means of the propagation of cholera. All that is claimed is that experience has proved that contaminated water-supplies have played a conspicuous part in the dissemination of the disease. On the other hand, the behaviour of cholera seems to require for its explanation a theory of the ability of the cholera organism to carry on certain of its life phases in the soil—a view which is now becoming generally held in this country with regard also to the organisms of enteric fever and diarrhœa. It is probably also true that the cholera organism is capable, under certain circumstances, of escaping from its habitat in the soil and infecting human beings, either directly or by fastening on to food. This almost certainly occurs in the case of the closely allied malady, diarrhœa.

It has been again and again observed that in England cholera has attained its widest diffusion during the second year of its appearance in the country; and in so far as the later diffusions were connected with the earlier appearances of the disease, it can hardly be doubted that, during the interval between the two, the cholera organism, although reduced to a relatively latent condition as regards pathogenic manifestation, must have continued its existence—presumably in the soil.

We are indebted to Mr. Power for the following hypotheses formulated by him in 1883–84 in provisional explanation of the general behaviour of cholera, and especially of the character of English cholera epidemics above referred to:—

CHOLERA HYPOTHESES, 1883

1. The cause of cholera is a living organism.

2. This organism has phases in its life-history, during some of which it has not, and in others it has, the power of producing cholera. Let these respectively be called 'non-malignant' phases and 'malignant' phases. In all phases the organism has the power of multiplying itself.

3. In most parts of the world the organism can exist in phases of both sorts. But only within certain geographical limits—its 'customary' area—has it the faculty of passing from non-malignant into malignant phase.

4. Alike in its non-malignant and in its malignant phase the organism is liable to be conveyed—in the body of a human host or otherwise—beyond its customary area, and be deposited here and there throughout the world.

5. Such convection of the organism in its non-malignant phase can never be demonstrable—i.e. by the appearance of cholera—inasmuch as *ex hypothesi* 3 it cannot, when existing outside its customary area, pass into its malignant phase.

---

[1] See also Dr. Snow, *On the Mode of Communication of Cholera*; Mr. Macnamara's *Treatise on Asiatic Cholera*; and various reports in the Eighth and Ninth Reports of the Medical Officer to the Privy Council (1885 and 1886).

6. But such convection of the organism when it is in its malignant phase can result in cholera, first, among the population immediately receiving it; secondly, among populations receiving it from that first population.

7. The resulting cholera in such case will appear only when local and climatic conditions are favourable, and the absence of cholera cannot, therefore, alone be evidence of the non-convection of the organism in its malignant phase.

8. The total of the local and climatic conditions required for the appearance of cholera in places outside its customary area, to which the cholera organism has been conveyed, is never continuous, but always recurrent or cyclical.

9. At a time when this total of conditions is present, the conveyed organism, being in its malignant phase, produces cholera. It produces cholera, however, only in quantity proportioned to its own quantity, or to the quantity of its immediate descendants produced during the maintenance of the requisite conditions. [Wherefore the local outbreaks of the disease are apt to be of small dimensions when they appear for the first time after the convection of cholera to a place.]

10. At a time subsequent to the original introduction into a place of the cholera organism in its malignant phase, the total of local and climatic conditions requisite for the appearance of cholera may be absent; and in that case the cholera organism, though in its malignant phase, can for a while await the reappearance of the required conditions. On their reappearance, cholera will appear as in § 9.

11. This waiting cannot be indefinitely prolonged, consistently with maintenance of the malignant phase among the descendants of the imported organism. If prolonged, the required local and climatic conditions can appear, but no cholera will appear.

12. After the appearance of cholera in a place, and when the local and climatic conditions required by the organism in its malignant phase are passing away, the descendants of the original organism will be, for a while, in existence, and they will be, in greater or less number, endowed with the same quality of malignancy as their ancestors.

13. From these immediate descendants, malignant and non-malignant, a succession of generations can be produced, the generation always tending towards the non-malignant phase of the organism.

14. Upon recurrence of the requisite local and climatic conditions, such organisms as have assumed the non-malignant phase cannot produce cholera.

15. Upon such recurrence, the organisms which continue to live in their malignant phase may or may not have become more numerous than in earlier generations. Often they have become more numerous. [Wherefore local outbreaks of cholera following upon a slight original outbreak (§ 9) are apt to be of larger dimensions than the original outbreak.]

16. The tendency of cholera organisms in their malignant phase, when existing outside their customary area, to die or to exhibit the non-malignant phase in their descendants, overcomes in the long run the tendency to multiply in any malignant phase. [Wherefore, unless exceptional conditions be present, the tendency of cholera is to disappear from localities outside its customary area.]

In connection with this subject, the remarks of Dr. Sims Woodhead upon cholera in his paper, already quoted, are of considerable interest. He points out that the cholera organism, when grown anaërobically, gains increased virulence, but largely loses its power of resistance to germicidal agents. Conversely, when grown aërobically it largely loses its poison-forming function, but gains in power of resistance. Its cultivation in the bodies of human hosts, therefore, while augmenting its virulence, does not tend to conserve that section of a given crop which has taken to colonise in the human subject. On the other hand, its aërobic existence outside the body, while diminishing its ability for immediate harm to human beings, increases its ability of maintaining itself (other essential conditions being present), and, consequently, its prospective danger to man when the season most favourable for its migration from the soil shall come round. In this way, also, Dr. Woodhead explains the fact that cholera displays but little tendency to spread immediately from person to person, notwithstanding that there is ample evidence of its dissemination by fomites, such as infected body-linen.

With respect to the subject of the spread of cholera, several other matters require mention. Most observers consider that the virus is given off

by the discharges from the sick, especially, according to some authorities, when those discharges are undergoing decomposition. To the recipient the virus would seem most usually to gain access by the alimentary canal, though it is not unlikely that it may be inhaled. The poison, as already seen, is without doubt in the main transported by human agency, but it is probably also, to some extent, air-borne, though only for short distances. An interesting case of the spread of cholera by milk has been recorded by Dr. Simpson, the Medical Officer of Health for Calcutta. In this case the milk was admittedly diluted with water contaminated with choleraic discharges.[1] Great and continued fatigue and alcoholic excesses are said to predispose to cholera.

Period of Incubation.—The period of incubation of cholera is usually quite short—from a few hours to three days; but, according to the late Dr. Parkes, it may be prolonged for ten or even twenty days.

Protection.—Cholera affords but a very slight degree of immunity, if any, against a subsequent attack.

Relation to other Diseases.—Cholera exhibits certain striking points of similarity to the epidemic diarrhœa of this country. In his report upon the latter disease Dr. Ballard remarks: 'The kinship of "diarrhœa" to malignant cholera is seen principally in the fact that both are clinically diarrhœal diseases in which the abundant watery discharges from the bowels are more or less rapidly succeeded by collapse, in which there is some similarity (in diarrhœal cases about to become fatal), in the *facies*, the sunken eyes, the shrinking of the bulk of the body, and algidity in certain cases. In both there is free desquamation of the intestinal epithelium; in both the kidneys are early implicated; and in both this condition may issue in uræmia and its results. Communicability through the medium of the morbid evacuations, although reputedly a character of malignant cholera, does not appear to be a character uniformly attaching to the disease; nor is non-communicability through the same medium a character invariably attaching to the epidemic diarrhœal malady.' Indeed, it would appear that, regarded from the clinical view point, such differences as there are between the two maladies are largely differences in degree of malignancy.

Etiologically, too, these diseases exhibit many points of likeness. Both appear to be associated with filth, and especially with excremental filth. Both seem to be very similarly influenced by season, and especially by heat. The physical conditions of soil favourable to the one disease appear to be precisely those which are favourable to the other, there being in each case much evidence to show that the prevalence of the disease is greater or less according to the permeability of the soil, and its consequent ability to harbour organic refuse.[2] As regards the degree of moisture of the soil favourable to the prevalence of these two diseases, there is again similarity—a moderate degree of moisture being favourable, and excessive wetness unfavourable, to both.

Lastly, while Dr. Ballard has shown that a close relationship subsists between the temperature of the soil and diarrhœa prevalence, a very similar relationship between the temperature of the soil and the spread of cholera seems to have been observed by Delbrück, and also by Pfeiffer, though their observations have not as yet been corroborated.[3]

[1] *Practitioner*, vol. xxxix. p. 144.

[2] Compare especially the evidence adduced by Ballard with respect to diarrhœa (Supplement to the Report to the Local Government Board, 1887), and Hirsch (*op. cit.*, vol. i. p. 452–3) for cholera.

[3] See Hirsch, *op cit.*, vol. i. p. 461.

From what has been said, it will have been seen that, both clinically and etiologically, diarrhœa and cholera have many points in common. It is, of course, generally held that they are absolutely distinct diseases; but there would seem at least some grounds for the surmise that cholera may after all be but an Asiatic variety of a disease known elsewhere as 'diarrhœa' and cholera nostras—a variety, that is to say, which has been reared during the course of time under conditions of environment especially suitable for the fostering of its malignancy. The more pronounced communicability of cholera, as compared with diarrhœa, is no sufficient objection to this view, for not only is it a fact that diarrhœa in England is also at times communicable, but the heightened malignancy which is postulated of cholera by the above hypothesis might reasonably be expected to carry with it an increase of communicability.

As an objection to the thesis here suggested, it might be urged that, according to experience in this country, contaminated water plays a far more conspicuous part in the dissemination of cholera than in that of epidemic diarrhœa. But with regard to this it has to be remembered, that while on the one hand epidemic diarrhœa in England does sometimes appear to be associated with contaminated water-supplies, on the other hand, it is by no means certain that in India the contamination of drinking-water is responsible for the same proportionate share in the diffusion of cholera as would appear to have been the case with respect to the English epidemics of that disease; indeed, if such were the case it would be difficult to understand the attitude taken up by many Anglo-Indian authorities with respect to this matter.

May it not rather be with cholera in India, as it would seem to be with diarrhœa in England, that in localities in which the disease is conspicuously endemic, the soil is so charged with the necessary microzyme [which, being indigenous to the locality, is capable of maintaining itself continuously and of 'cropping' year by year] that the disease, although doubtless even there disseminated from time to time by water, is also largely, and perhaps more largely, disseminated by direct emanation from the soil—that in India, in other words, the spread of cholera is an affair more after the kind of that of diarrhœa in England? But that when the cholera microzyme is carried into other lands, and more especially into countries within the temperate zone, where the conditions are less favourable to its rapid multiplication in the soil, its opportunities for infecting man, particularly during the first season of its importation, are more largely dependent upon those chance occasions upon which it is conveyed to him in infected articles of clothing or of commerce, contaminated water-supplies, and the like? Such a view might, perhaps, afford a basis for reconciliation between the Anglo-Indian and British schools with respect to the etiology of cholera.

In closing these remarks upon cholera, one note of warning may be sounded. It must not for a moment be concluded that, because England has escaped cholera since 1866, she will enjoy a permanent immunity from that disease. It is true, of course, that of late years very considerable improvement in the sanitary condition of the towns has taken place, and, other things being equal, such improvement may doubtless be relied upon as meaning an equivalent diminution in the liability of cholera to spread. Nevertheless, there are still many localities in this country in which, owing to the supineness, in the past, of the Sanitary Authorities presiding over them, cholera would most undoubtedly cause havoc if it should unhappily gain access to them. Moreover, we cannot count upon 'other things being equal.' There is cholera and cholera, and the chance of its obtaining a foothold with

us must doubtless largely depend upon the amount and the type of the cholera sent to our shores. To illustrate the point, it may be mentioned that any person who, in view of our prolonged immunity from influenza, had inferred our permanent freedom from that malady, would during the last three years have had a somewhat rude awakening. Of course there are vast differences between influenza and cholera, but the biological factor has to be reckoned with as regards both, though no doubt it counts for less in respect to the spread of the latter than the former disease.

Neither must any abatement of cholera in Europe during the winter of 1892–93 be taken as meaning the end of our danger. On the contrary, reference to the behaviour of European cholera in the past gives abundant grounds for anticipating that it will almost certainly reappear upon the Continent in the spring, and attain, probably, a far wider prevalence there than it has done during the summer of 1892. If such should unfortunately turn out to be the case, we shall have to defend ourselves against imported cholera from a number of different ports, and for a period of many months. And it must be remembered, then, that no system of quarantine or of medical inspection can be safely relied upon to keep out the disease, and that the only true safeguard is to be found in pure water-supplies, efficient methods of excrement and refuse disposal, and wholesome conditions of life generally.

EPIDEMIC DIARRHŒA

Synon.: *Choleraic Diarrhœa; English Cholera; Cholera Nostras; Infantile Cholera; Muco-enteritis; Gastro-enteritis; Dysenteric Diarrhœa; Alvus soluta; Defluxio.* Fr. *Diarrhée, Cours de ventre*; Ger. *Durchfall*; It. *Diarrea.*

Diarrhœa, in the sense of a mere flowing from the bowels, may be simply a physiological process—the natural reaction of the healthy bowel against obnoxious contents. Again, it may be due to nervous influences, or be symptomatic of various morbid conditions of one or other of the internal organs; and sometimes it is compensatory, or 'vicarious.' But there are now abundant grounds for the view that the diarrhœa which so conspicuously swells the mortality returns is of quite another sort, and, indeed, belongs essentially to the category of epidemic diseases. This has for some years been recognised by the Registrar-General, who includes diarrhœa among his 'Principal Zymotic Diseases.' The Royal College of Physicians also classifies 'Epidemic Diarrhœa' along with 'Specific Febrile Diseases.'

This view of the nature of diarrhœa was based in the first instance upon the broad analogies between diarrhœa, regarded in a comprehensive manner, and other epidemic diseases. Thus, diarrhœa affects large numbers of persons at the same time and place; it displays a very decided affinity for certain populous localities (indeed, so marked is this circumstance as to have led the Registrar-General to frequently refer to certain towns as diarrhœal towns); and it is influenced in a remarkable degree by season.[1]

But the more recent researches of Drs. Ballard and Klein have now established the title of diarrhœa to be regarded as an epidemic disease. Apart from

[1] The seasonal rise in the diarrhœa mortality, inasmuch as it occurs during the height of the summer, has often been attributed to the eating of fruit. This, however, can hardly be the true explanation, since the mortality from summer diarrhœa falls mainly upon the children under one year of age. Such children do not, as Dr. Longstaff remarks, eat extensively of fruit, and this cause could therefore only be thought of as

the more detailed points of agreement between it and other epidemic diseases, in the matter of epidemiological features, which may be gathered from the summary, about to follow, of Dr. Ballard's Report,[1] these observers have conclusively shown that diarrhœa is marked out, both by its symptoms and pathology, as a general disease, of which the diarrhœa is but one of its several manifestations. In addition to certain changes in the alimentary canal which might have been anticipated, decided changes were invariably found after death in other organs, notably the kidneys, liver, and lungs. Indeed, Dr. Ballard remarks that 'in the bodies examined there were marked pathological changes, not only in the intestines, but in all the viscera; and not alone in the viscera of protracted cases, but in those of infants the total duration of whose illness had not exceeded twelve or fourteen hours.'

In studying the symptomatology of diarrhœa, Dr. Ballard and Mr. Power personally inquired into 340 fatal cases at Leicester during the summers of 1881 and 1882.

The 'leading phenomena' of the disease Dr. Ballard describes as 'diarrhœa, vomiting, convulsive phenomena; a bodily temperature at certain periods above, at other periods below what is normal; reduction in quantity or actual suppression of urine, embarrassed breathing, and, where looked for, commonly physical indications of pulmonary hyperæmia or inflammation, pallor of surface of the body, loss of bulk and flesh, and exhaustion, with its various well-known clinical features. I must add, that occasionally there is jaundice. Now and then a (fugitive) rash has been observed on the body.'

Dr. Ballard's more detailed remarks upon these symptoms should be studied in his report. It may, however, here be mentioned that, while stating that diarrhœa is most frequently 'the predominant symptom,' he says: 'I may here state my strong suspicion, almost my belief, that the malady usually characterised by diarrhœa may run its course from first to last, and even to death, without any remarkable diarrhœa at all. In other cases, although diarrhœa occurs, it is by no means the prominent symptom of the disorder; it may be comparatively of trifling amount or of short duration.' As regards vomiting, he points out that 'in only 43 out of 326 fatal cases occurring in Leicester of which I have notes (and in which the presence or absence of this symptom is mentioned) was vomiting absent altogether.'

The vomiting usually occurs coincidently with the diarrhœa, but 'occasionally it stands alone, perhaps for a whole day, as the prominent feature of the attack.' 'As a rule, the shorter and sharper the illness, the earlier in its course does this symptom appear.' Convulsions were present at some period or other of the attack in all but a small percentage of these fatal cases. They were mostly regular 'fits,' but in about a fifth of the cases they consisted of one or other of the well-known minor convulsive phenomena of children. With respect to the convulsions, Dr. Ballard remarks that 'there is reason to regard them not only as one of the most important phenomena of the malady, but, when occurring, as they mostly do occur, late in the illness, as indicating (and probably then as often due to) an uræmic condi-

operating in a wholesale way, 'through the maternal organism, which is possible, though not in a high degree probable.' Another objection to the theory is, that in the rural districts, where the eating of fruit might be expected to be at a maximum, the diarrhœa mortality is at a minimum. Similarly, the increased summer mortality has been attributed solely to atmospheric heat. It was, however, some time ago pointed out by Sir George Buchanan that a high temperature during the second quarter of the year does not give rise to the typical annual epidemic, and, as will be seen later, it has now been shown by Dr. Ballard that atmospheric heat alone does not suffice as a cause of the increased mortality.

[1] Supplement in continuation of the Report of the Medical Officer of the Local Government Board for 1887.

tion, the result of the kidney affection invariably present in these cases. The *comatose condition* in which the patient often dies appears also as a rule due to the same condition of the blood; but in some cases evidences of intra-cranial inflammation have been observed.'

In referring to the temperature charts published with his report, Dr. Ballard says: 'One important fact which they indicate, so far as my inquiry is concerned, is the "algide" character of the developed malady. At the commencement there appears to be some little febrile disturbance; but sooner or later, in the cases about to become fatal, the temperature falls more or less below the normal range, being lower in the morning than the evening, and even then it mostly fails to attain a normal standard. Towards the end of a fatal case the temperature is apt to rise. . . . In several cases of very short duration, where the child is rapidly prostrated with deluging watery stools, the temperature falls rapidly, and the child may die in collapse.'

In touching upon the subject of symptoms, we have gone somewhat beyond the scope and intention of these papers, but it seemed necessary to do so in the present instance, as an essential part of the argument for the right of diarrhœa to rank as a general disease.

With respect to the history and geographical description of diarrhœa little need be said. The malady is clearly described by Hippocrates, and seems to have been more or less present in all times and in all places. It has been pointed out by Sir George Buchanan, however, that, in its conspicuous epidemic form, diarrhœa appears, so far as this country is concerned, to be a comparatively modern phenomenon—at least, judging from the old Bills of Mortality.

Mortality.—The English mortality from 'diarrhœa and dysentery' year by year from the commencement of registration will be found in the following table, compiled from the Registrar-General's Annual Reports:—

TABLE XII.—*England and Wales.—Deaths from Diarrhœa and Dysentery.*

Year	Deaths	Rates per million living	Year	Deaths	Rates per million living
1838	3,109	203	1865	24,603	1,164
1839	3,099	200	1866	18,266	853
1840	4,097	260	1867	20,813	960
1841	3,755	236	1868	30,929	1,405
1842	6,002	372	1869	20,775	935
1843	—	—	1870	26,126	1,161
1844	—	—	1871	24,937	1,094
1845	—	—	1872	23,034	995
1846	—	—	1873	22,514	962
1847	14,842	863	1874	21,888	923
1848	13,696	787	1875	24,729	1,028
1849	20,881	1,189	1876	22,417	917
1850	13,504	760	1877	15,282	619
1851	16,913	941	1878	25,103	1,003
1852	20,373	1,117	1879	11,463	452
1853	16,083	874	1880	30,185	1,171
1854	21,995	1,181	1881	14,536	558
1855	14,207	754	1882	17,185	653
1856	15,150	793	1883	15,933	598
1857	22,887	1,189	1884	26,412	978
1858	15,331	787	1885	13,398	492
1859	19,710	1,001	1886	24,748	899
1860	10,858	546	1887	20,242	727
1861	20,162	1,002	1888	12,839	455
1862	12,156	597	1889	18,434	648
1863	15,994	775	1890	17,429	606
1864	17,432	832	1891	13,653	469

In connection with fatal diarrhœa, Dr. Ballard's report deals with two important and interesting matters: 1. The duration of the malady in fatal cases; 2. The influence exerted upon the duration of the fatal illness by previous healthiness or weakliness.

1. It appears that the malady may be fatal in a few hours, or may linger for three, four, or even six or eight weeks. Of the 340 fatal cases observed at Leicester in the epidemic periods of the years 1881 and 1882, about one-half were fatal in less than a week; about one-quarter in between one and two weeks; between an eighth and ninth before the end of the third week; and about 15 per cent. at some time later than this. A further study of these fatal cases shows that the 'ferocity' of the malady, as judged by the shortness of the illness, increases with progress of the epidemic—at least, it was so at Leicester in the years 1881 and 1882. The increase, however, was not altogether a steady increase, but seemed inclined to occur in waves, and there were differences in the increase of the two years.

2. The previous health of 332 out of the 340 Leicester cases was ascertained. Of these 332, 141, or 42·5 per cent., were recorded healthy, and 191, or 57·5 per cent., either as weakly from birth or for a longer or shorter time prior to their fatal diarrhœal illness. It would therefore seem that '*infantile diarrhœal mortality is in part dependent upon previous general health.*' In connection with age in these cases, the figures showed that the influence of previous weakly condition on mortality was most conspicuous under nine months of age. It was also found that previous weakliness had '*much to do with the* speedy collapse of the *most rapidly fatal cases* and *with the cases which have a duration of forty-eight hours and under four days, as also with the fatal cases of the most prolonged duration*—i.e. three weeks and upwards.'

But when the proportion of weakly to healthy children fatally attacked at different periods of the epidemic seasons of 1881 and 1882 came to be studied, it was found that the weakly were by no means specially attacked during the early periods. On the contrary, the evidence went rather to indicate that 'weakly children require a longer exposure to the epidemic cause, whatever that may be, than healthy children.'

Influence of Sex.—The incidence of the disease appears to be greater upon children of the male sex at all ages from birth upwards. The mortality is greater for males in infancy and old age, but usually rather greater for females from the second or third to about the fiftieth year. At all ages together it is greater for males, doubtless owing to the larger number of males living during the early period of life.

Influence of Age.—As regards age in relation to *attack*, Dr. Ballard finds that 'the actual incidence upon age (by which term wherever it is used is signified relation to the population living at the several ages) is chiefly upon children under five years of age, and of these most upon those under two years.

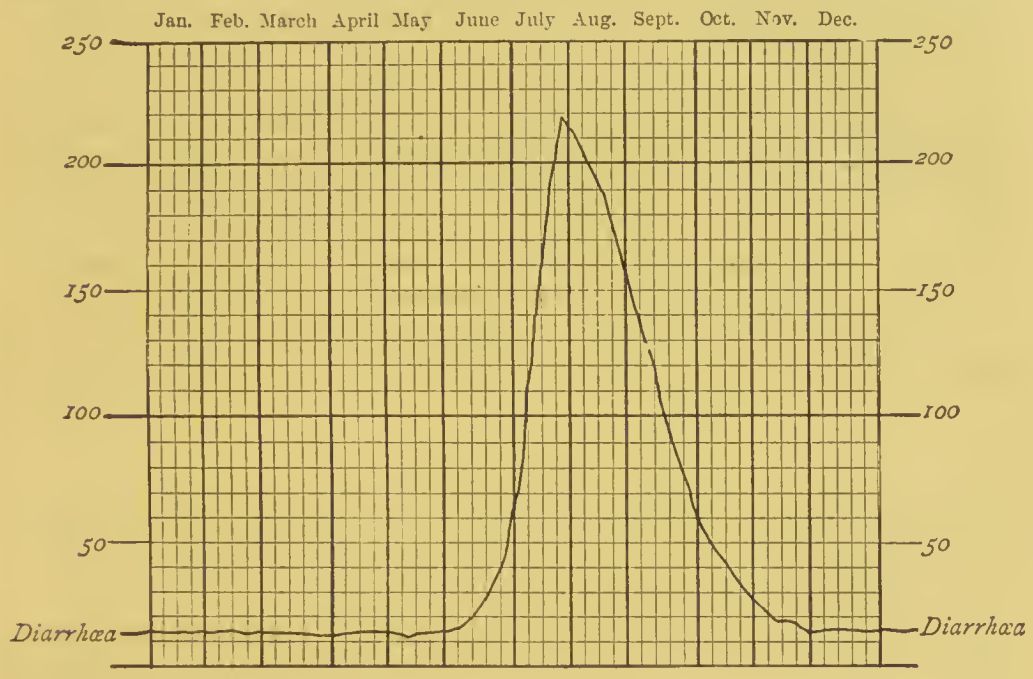
'After five years of age the actual incidence lessens remarkably, and is least between the ages of five and twenty-five years, after which it gradually increases.

'Among children under five years of age the incidence is comparatively small upon those under three months, after which age it apparently increases; beginning to lessen somewhere between the ages 1–2 years.' The incidence upon age, however, varies in different years, and it appears that 'in years of greater general prevalence the increased prevalence is more obvious, on the whole, upon those over five years of age than upon those under five years.'

The large majority of the diarrhœa deaths occurs during the first year of

life, the second three months of that year having the highest mortality. After the second year the deaths are few, and gradually diminish until the twentieth year, when they again steadily rise throughout the remainder of life.

Influence of Season.—The influence of season on London diarrhœa mortality is shown in the curve here given, which is taken from the Registrar-General in his Annual Summary for 1890. The curve represents the actual average number of deaths each week, and is based on the diarrhœa deaths



registered in London during the fifty years, 1841 to 1890. It appears from the curve that the conspicuous rise in the London mortality corresponds in point of time with the conspicuous rise in prevalence as shown in a case-table given by Dr. Ballard, and the maximum is attained in the same four-weeks period.

Cause and Dissemination.—The provisional results of Dr. Ballard's inquiry into the causation of diarrhœa may be summarised as follows :—

A. General Conditions

Atmospheric Temperature.—A high atmospheric temperature conduces to diarrhœal mortality, but it operates *indirectly*, and is not the main cause.

Temperature of the Earth.—This is a far more important condition. Dr. Ballard 'made for London and many other towns in the kingdom a large number of charts, showing week by week for many years the earth temperature at a depth of one foot from the surface and at a depth of four feet from the surface, each chart showing also the diarrhœal mortality of the corresponding weeks.'

The general results shown by these charts are stated as follows :—

'*a.* The summer rise of diarrhœal mortality does not commence until the mean temperature recorded by the 4-foot earth thermometer has attained somewhere about 56° Fahr., no matter what may have been the temperature previously attained by the atmosphere or recorded by the 1-foot earth thermometer.

'*b*. The maximum diarrhœal mortality of the year is usually observed in the week in which the temperature recorded by the 4-foot earth thermometer attains its mean weekly maximum.

'*c*. The decline of the diarrhœal mortality is in this connection not less instructive, perhaps more so, than its rise. It coincides with the decline of the temperature recorded by the 4-foot earth thermometer, which temperature *declines* very much more slowly than the atmospheric temperature, or than that recorded by the 1-foot earth thermometer; so that the epidemic mortality may continue (although declining) long after the last-mentioned temperatures have fallen greatly, and may extend some way into the fourth quarter of the year.

'*d*. I do not wish it to be inferred that the atmospheric temperature, and the temperature of the more superficial layers of the earth, exert no influence on diarrhœa. Their influence, however, is little, if at all, apparent until the temperature recorded by the 4-foot earth thermometer has risen as stated above. Then their influence is apparent, but it is a subsidiary one.'

Rainfall exerts an influence upon diarrhœal mortality, but probably an indirect one, through its influence on the temperature of the soil.

Air-movement also influences diarrhœal mortality, wind lessening, and calm, in the diarrhœal season, promoting it.

B. Conditions of Locality

Elevation above sea-level has some influence, but by no means a controlling one. Indeed, it seems to influence diarrhœal mortality 'only in so far as it affects infant mortality from all causes together.'

Soil seems to have a decided influence, and this is, apparently, largely a matter of porosity. Of whatever nature the soil, the more loose and porous it is, the more conducive to diarrhœal mortality. Hence, sand and mould most favour diarrhœa. Where the houses are built upon solid rock the diarrhœa mortality is low. Organic fouling of the soil, whether excremental or vegetable, is distinctly favourable to a high diarrhœal mortality. Persons, therefore, living upon soil polluted by leaking sewers, drains, or cesspools, or upon 'made soil,' or the site of market-gardens, are apt to suffer from a high diarrhœal mortality. Even persons living upon rock are liable to suffer more from diarrhœa if the rock is much fissured than if such is not the case, owing apparently to the opportunities for the accumulation of filth in the fissures.

Both excessive wetness and excessive dryness of soil seem to lessen diarrhœal mortality, but a moderate dampness of soil favours it—i.e. 'an amount of habitual dampness which is decided, although not sufficient to preclude the free admission of air between the constituent physical elements of the soil.'

Density of population favours, and the reverse condition disfavours, diarrhœal mortality. This is seen by a general comparison of the 'diarrhœal' towns of various sizes, and by a comparison of that of the more and less crowded parts of the same towns. The influence of aggregation, although observable upon infant mortality from other causes, is most noticeable with respect to diarrhœa. It is, however, probably less direct than in the case of measles or scarlet fever.

Density of buildings (*whether dwelling-houses or others*) *upon area*, as in factory towns, appears to favour diarrhœal mortality, and 'it is probable that this difference of density of buildings upon area is one of the circumstances which have to do with the difference of diarrhœal mortality between large and small towns.'

Restrictions of, and impediments to, the free circulation of air, whether about or within dwellings, promote diarrhœal mortality. As an instance of the latter case, 'back-to-back' houses are particularly objectionable.

Domestic darkness and general dirtiness within dwellings, as well as filthy accumulations of domestic refuse in privies, ashpits, dustbins, &c., are conducive to diarrhœal mortality.

Sewer and cesspool emanations, 'especially in a concentrated form and suddenly let loose, may occasion attacks of fatal diarrhœa.' They may, probably, also cause a diarrhœal epidemic in a non-diarrhœal season, and hence in all likelihood operate in a similar fashion in the diarrhœa season.

Mere coal smoke, and emanations from chemical works, seem without special influence.

Undefined polluted conditions of drinking-water at times seem to cause epidemic diarrhœa, but there is at present no evidence that water-pollution plays a prominent part in the causation of the annual infantile diarrhœa mortality of this country.

C. Conditions Relating to Population

Social Position.—Diarrhœa mortality is notoriously greatest among the lower classes. This, however, is the case, and even to a greater extent, with mortality from other causes.

Food, no doubt, is concerned with the prevalence of epidemic diarrhœa, though probably not, as generally supposed, by reason of its causing ordinary dyspepsia, but owing to its contamination with some substance, 'which substance is by itself an efficient cause of the malady.'

a. As regards the influence of the mode of feeding young infants. This was especially studied for Dr. Ballard by Dr. Hope, the Assistant Medical Officer of Health for Liverpool, the general result of whose inquiries closely coincide with previous medical opinion on the subject. They are expressed by Dr. Ballard as follows:—

'1. That infants fed solely from the breast are remarkably exempt from fatal diarrhœa, even among the low-class Irish, the degree of exemption being exactly the same among the Irish as among the English and other races in his city.

'2. That infants fed in whatever way with artificial food, to the exclusion of breast-milk, are those which suffer most heavily from fatal diarrhœa.

'3. That children fed partially at the breast, and partially with other kinds of food, suffer to a considerable extent from fatal diarrhœa, but very much less than those who are brought up altogether by hand.

'4. As respects the use of "the bottle," that it is decidedly more dangerous than artificial feeding without the use of the bottle.'

b. Food stored in dark, ill-ventilated places, and exposed to telluric or other unwholesome emanations, probably becomes liable to cause diarrhœa.

Maternal neglect conduces to infant mortality from diarrhœa. Thus, the mortality among illegitimate infants is greater than that among the legitimate from all causes, but the increase is rather greater in the case of diarrhœal mortality than of that from other causes. This, however, is only observable in years of low epidemicity—'the presumably less potent or less abundant specific cause (in such years) operates fatally more easily on the illegitimate than on the better cared-for class of infants.' But in highly epidemic years the illegitimate seem to be the earliest to suffer.

The occupation of females from home, as conducing to neglect and artificial feeding of infants, promotes diarrhœal mortality.

The propositions provisionally formulated by Dr. Ballard with respect to diarrhœa causation will be found on page 366, vol. i., and therefore need not be repeated here.

Diarrhœa is sometimes highly infectious, the infection apparently being associated with the bowel discharges of the patients. An account, by Dr. R. Bruce Low, of four particularly interesting and instructive outbreaks of communicable diarrhœa of this sort will be found in the appendix to Dr. Ballard's report.

Relation to other Diseases.—The question as to the kinship of diarrhœa and cholera has been considered in the section on the latter disease.

The analogies between diarrhœa and scarlet fever are interesting, and deserving of careful attention and study. They are thus referred to by Dr. Ballard:—

'As in scarlet fever, so in "diarrhœa," the condition of the kidneys is an essential element in the disorder, and the uræmia observable in the one disease finds its analogue in the other. From this view-point the intestinal lesions in diarrhœa may be but the analogue of the skin eruption in scarlatina. That the one disease is notoriously communicable from one person to another, while the other is usually regarded as non-communicable, goes for little as an argument against such analogy, since, on the one hand, very many outbreaks of scarlatina (namely, those of milk origin) seem to have hardly any such communicable quality, and, on the other hand, communicability is a quality not unknown among cases of epidemic diarrhœa. In both diseases it is from the cutaneous or quasi-cutaneous (mucous) surface chiefly implicated that the contagious principle gets abroad—viz. the skin and throat surfaces in scarlatina, and the intestinal mucous surface in "diarrhœa."'.

The question of a possible relationship between diarrhœa and enteric fever needs also to be kept in view.

Period of Incubation.—Judging from Dr. Bruce Low's cases of communicable diarrhœa, the period of incubation appears to be very short—on an average, ten to twelve hours.

MALARIAL DISEASE

Synon.: *Ague; Intermittent Fever; Remittent Fever; Bilious Remittent Fever; Paludal Fever; Miasmatic Fever.* Fr. *Fièvre Aiguë, Fièvre Paludéenne, Fièvre Rémittente*; Ger. *Wechselfieber, Bösartiges endemisches Fieber*; Ital. *Febbre Intermittente, Febbre Remittente.*

Malarial disease, being intimately associated with conditions of soil, has already been dealt with as regards its etiology and the measures necessary for its prevention, by Dr. Copeman, in the first volume of this work. As regards bacteriology, it has also been dealt with by Dr. Klein. There are, however, still certain points of interest which may be referred to here.

It will be observed, of course, that the synonyms given above do not all apply to clinically identical manifestations, and it is regarded by some as not unlikely that bacteriology may ultimately establish 'specific' distinctions between the conditions represented by certain of them, such as intermittent and remittent fever. Indeed, functional and even slight morphological differences are already said to have been observed among the microbes which

appear to stand in causal relation to the various forms of malarial fever. It must, however, be remembered that such differences, instead of indicating 'specific' distinctions among the micro-organisms in question, may but represent different phases, normal or developmental, in the life-cycle of one and the same species of organism; and that such is actually the case seems to be the inference suggested by epidemiological evidence. The several forms of malarial disease are, therefore, here regarded as varieties of the same malady, differing mainly in degree of malignancy.

As regards history, clear record of malarial illness is found in the medical writings of antiquity, but the fuller accounts of the disease seem to date from the sixteenth century. In its distribution malarial disease still covers an enormous area of the world's surface, though it is far more prevalent and intense in the tropical and sub-tropical than in the temperate countries; and it does not, apparently, extend at all beyond the limits of 63° N. and 57° S. 'Covering a broad zone on both sides of the equator, the malarial diseases reach their maximum of frequency in tropical and sub-tropical regions. They continue to be endemic for some distance into the temperate zone, with diminishing severity and frequency towards the higher latitudes. . . .'[1]

Space does not permit of a detailed account of the distribution of malaria being given here, and for particulars on this point the reader must be referred to Hirsch and other authorities. The main features of its present distribution, however, seem to be as follow:—It is widely prevalent, and in a virulent form, in tropical Africa, especially on the west coast. It is also prevalent in Algiers and in parts of Egypt, as the Nile valley. In Asia it is conspicuously present throughout India, Ceylon, China, Afghanistan, Persia, Arabia, and Syria; in the Western Hemisphere, the West Indies, Brazil, Peru, the coast of the Gulf of Mexico, and the southern and central parts of the United States. In Europe it is most severely prevalent in Italy, but occurs to a greater or less extent in every country except the Faröe Islands and Iceland. Great Britain in the present day suffers but little, though the disease still lingers in the counties on the east coast—notably in the fen districts of Lincolnshire, Norfolk, Huntingdon, and Cambridge; but even in these localities it has of late almost disappeared. In Ireland it is but very slightly present, and Scotland is almost, if not entirely, free from it.

Although, as has been said, malaria is most prevalent and most malignant in tropical and sub-tropical countries, yet among such countries certain districts are far more malarious than others, while some enjoy a complete exemption in this respect. As regards India, for instance, the Presidency of Madras suffers much less than those of Bengal and Bombay. On the West Coast of Africa, according to Hirsch, malaria becomes less severe from Cape Lopez southwards, and this exemption 'becomes more and more marked the nearer we approach the Cape of Good Hope, which itself enjoys, along with St. Helena, an almost complete immunity from the endemic fever.' For instances of other important immunities, New Zealand and Tasmania are said to be completely, and Australia almost completely, exempt.

As might be expected of a malady presumably caused by micro-organisms that are largely saprophytic, malarial disease is, perhaps, more than almost any other disease of the class under consideration, entitled to be described as 'endemic.' Yet it is a point of great interest that even this disease has at times exhibited decided epidemicity, extending to localities in which it is not usually met with. This circumstance raises the question whether in such epidemic extension there is actual transport of the malarial organism beyond its ordinary endemic areas, or whether the exceptional appearance of the disease

[1] Hirsch, *op. cit.*, i. 197.

in localities not ordinarily prone to it may not be consequent upon the acquirement of pathogenicity there by organisms which are usually benign—as a result, of course, of exceptional conditions, meteorological or other. Or a third hypothesis might be suggested, to the effect that the exceptional conditions referred to operate indirectly by increasing the susceptibility of individuals, and thus rendering them vulnerable to attack by organisms, indigenous to the neighbourhood, but not usually pathogenic. The fact, however, that some districts, which up to a certain time have enjoyed immunity from malarial disease, such as Réunion and Mauritius, have subsequently become endemic centres of that malady, might be quoted as suggestive of the transportation of the organism. It seems to have been shown by Dr. Salisbury that the malaria organism may be transported from place to place in soil; and Sawyer records that while living in a malarious part of Illinois he 'visited a friend at Milton, Mass., and fell ill of intermittent fever. The lady of the house, who interested herself greatly on the patient's account, and who had never seen a case of ague before, had a slight aguish attack of fever and chill on the fifth day, with gastric disturbance; but she set aside the idea that she could possibly be ill of ague, as the disease was quite unknown in Milton, or occurred only now and then in imported cases. However, she had a more severe attack the day after, and on the ninth day the first pronounced paroxysm of ague occurred, and with that all doubt as to the nature of the disease vanished.'[1] This case is at least suggestive of the conveyance of malaria in clothing, or even of direct infection from case to case. And the fact that malarial disease did not become prevalent in Réunion until some three years after it had been epidemic in Mauritius, is consistent with its having been imported there by persons or things from the latter place. The large majority of observers have, of course, declared against the spread of malarial disease by infection, and it may, no doubt, be concluded that such a mode of dissemination is at least not the rule; but it is by no means certain that it never occurs. In studying the subject in future, opportunity should be taken of observing the behaviour of the disease in this respect when it occurs in epidemic form, especially beyond the limits of its endemic area, as it would probably be under such circumstances that its infectiousness, if it exists at all, would most show itself.

As regards the distribution of the various forms of malarial disease, the more severe kinds—e.g. the remittents—are most common in those tropical and sub-tropical districts in which malarial disease generally is most conspicuously endemic, the disease in temperate climates being more commonly of the less severe intermittent type. And the same seems for the most part to hold good as regards the sub-varieties of intermittent fever, the severer forms with the short intermissions, such as the quotidian, forming a larger proportion of the cases in warm than colder countries, and *vice versâ*. With respect to variations in type from time to time, it appears, according to Hirsch, that 'at the beginning of the epidemic, or the rise of the endemic, intermittent fevers are observed almost exclusively; that in the subsequent progress the cases of severe sickness become more and more numerous, predominating at the height of the epidemic, again becoming relatively fewer as the amount of sickness decreases; while only intermittent forms are observed at the close.'

This is highly suggestive of the progressive development of infectiousness, already referred to in connection with certain other epidemic diseases, and is very similar to the rise and fall of malignancy described by Dr. Whitelegge as characteristic of the true developmental cycles of measles and scarlet fever.

[1] *Boston Medical and Surgical Journ.*, Dec. 1867, p. 538. Quoted by Hirsch.

In several other respects, also, the phenomena of malarial disease are of special interest as regards their possible bearing upon analogous phenomena in connection with other diseases. The chronic character, for instance, of malarial disease, as evidenced by the so-called malarial 'cachexia,' and the fact that persons who have once suffered from intermittent fever are liable to recurrent attacks years after they have left a malarious neighbourhood, seems to have its analogue in the cases occasionally met with of chronic and recurrent diphtheria. The facts, too, with regard to protection or acquired immunity in malarial disease are of the highest interest, and deserving of special study in connection with the subject of protection generally. For here we have a disease in which repeated attacks are common, and, indeed, in which one attack may in a sense be said to increase liability to another, and yet, paradoxical as it may appear, there is evidence that the disease does, to some extent, at least, protect against itself. The answer to this seeming contradiction may possibly be, that while one attack increases liability to another attack, i.e. a recrudescence, it nevertheless protects against a *re-infection*. The negro race suffers decidedly less from malaria than most, and probably all, other races of mankind, but that their immunity is due to prolonged exposure to the poison is suggested by the alleged fact that negroes after long residence in non-malarious districts have, upon subsequent exposure to malaria, been readily attacked. As regards Europeans also, continued residence in malarious countries seems to confer protection against the more severe forms of the disease. On the other hand, it is the severe remittent form that, in unhealthy districts, frequently attacks new-comers. Lastly, while it is notorious that repeated attacks of the milder intermittent form of the disease are frequent, second attacks of the more severe remittent form, according to Maclean, less often occur, and persons who have suffered from remittent fever shortly after their arrival in a malarious country 'are seldom exposed to second attacks' [of remittent fever].

In the above facts we may, perhaps, see illustration of the establishment and repeated renewal of protection by small doses of the virus, and also evidence of relation between the permanency of protection and the dose or quality of the virus.

As regards sex- and age-influence, males appear to suffer more frequently than females, but this may be due to increased opportunities for infection. No age is exempt, but attacks are least frequent among the very young and the old. Sir Thomas Watson, however, quotes a case from Dr. Russell's 'History of Aleppo,' in which a 'woman had tertian ague, which attacked her, of course, every other day, but on the alternate days, when she was well and free, she felt the child [*in utero*] shake; so that they both had tertian ague, only their paroxysms happened on alternate days.'

Although in localities in which malaria is endemic the disease occurs in any season of the year, its prevalence yet appears everywhere to be largely regulated by season, though the particular time of year in which it is most common varies in different climates. Thus, in temperate regions, according to Hirsch, it seems to have two maxima, one in the spring, and another in the autumn, the latter one being the more important. In the warmer climates, where malaria is more rife, the maximum is in the late summer and early autumn, the spring maximum of the temperate climate practically disappearing; lastly, in the most malarious districts in the tropics the maximum prevalence is during and towards the close of the rainy season.

Malaria may, without doubt, be conveyed by air-currents, but for what distances is uncertain. Parkes considered that the distance is seldom greater

than one or two miles, and that it was to be doubted whether 'belief in transference of malaria by air-currents for ten, twenty, or even 100 miles is correct.' In any case, its conveyance by air is, to a large extent, arrested by belts of trees and sheets of water. On the other hand, 'when favoured by ravines and currents of heated air, it can scale mountains to a height which appears to differ in different climates, varying from four or five hundred to two or three thousand feet' (Maclean: Quain's 'Dictionary of Medicine').

The evidence with regard to the spread of malaria by water is conflicting. There is, no doubt, a very strong belief in this mode of its transmission among the inhabitants of malarious districts, and there certainly would seem to be some evidence in support of the view. Probably the strongest case is that recorded by Boudin. In this case 120 soldiers embarked in the 'Argo' for transport from Bona, in Algiers, to Marseilles. During the voyage 111 of them, thirteen of whom died, suffered from different forms of malarial fever. Two other vessels, carrying between them 680 soldiers, also from Bona, and arriving at Marseilles the same day as the 'Argo,' had no cases of illness at all, and the only ascertainable difference of circumstance between the troops in these ships and those in the 'Argo' was the difference of drinking-water. The latter were exceptionally supplied with water, which was said to have an unpleasant smell and taste, from a marsh near Bona; those on the other ship were supplied with good water. Finally, the nine soldiers on the 'Argo' who escaped were said to have purchased wholesome water from the crew of that vessel.[1]

The incubation period of malaria would seem, according to the evidence, to vary considerably, but more extensive data are required. Fagge gives it as usually from six to twenty days, but quotes cases recorded by Maclean and Hertz, of Amsterdam, which seem to show that it may be as short as a few hours.

ERYSIPELAS

Synon.: *St. Anthony's Fire; The Rose.* Fr. *Érysipèle*; Ger. *Erysipelas, Rothlauf*; It. *Risipola.*

History and Geographical Distribution.—The term erysipelas, as applied to spreading inflammations of the skin accompanied by fever, is found in the earliest medical records; and, indeed, the distinction between erysipelas occurring in association with wounds and erysipelas (regarded as) occurring independently of wounds, which has survived to the present day, dates back to the writings of Hippocrates.

From time to time, however, opinions have differed widely as to the actual processes which should be embraced by the term erysipelas; whether, for instance, it should include erythema, and certain inflammatory conditions of the mucous linings of the throat, lungs, and pelvic viscera; whether, also, it is to embrace the suppurative inflammations of the skin and subcutaneous cellular tissue, known in this country as 'phlegmonous erysipelas'—a point contested by Volkmann and other German authorities.

Unfortunately, the precise knowledge required for definitely setting at rest some of these questions is still wanting, as will appear later on.

As regards geographical distribution, erysipelas is tolerably uniform in its distribution throughout the temperate zone of both hemispheres. It also

[1] For further evidence on the subject see vol. i. p. 272 of this work.

occurs with tolerable frequency in cold countries, as Iceland, the Faröe Islands, and Greenland, and is not uncommon in warm sub-tropical latitudes. It seems, however, to be of decidedly less frequent occurrence in the tropics, though there are accounts of it at Réunion; and in the traumatic form, at least, it is met with in India.

Mortality.—In the following table will be found the number of deaths recorded year by year in England and Wales as due to erysipelas for the years 1838 to 1891, exclusive of the four years, 1843 to 1846, for which period the causes of death were not abstracted by the Registrar-General.

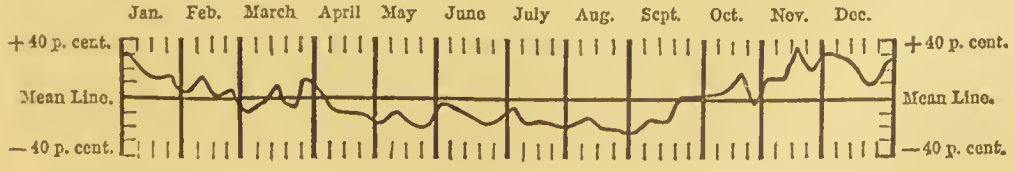
TABLE XIII.

England and Wales							
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1838	1,203	81		1866	1,665	78	
1839	1,140	75		1867	1,446	69	
1840	1,217	79	76	1868	1,943	88	82
1841	1,139	73		1869	1,854	83	
1842	1,111	70		1870	2,120	94	
1843	—	—		1871	2,216	97	
1844	—	—		1872	1,771	76	
1845	—	—		1873	2,027	87	106
1846	—	—		1874	3,358	142	
1847	2,050	—		1875	3,018	126	
1848	2,618	—		1876	2,327	95	
1849	2,303	—		1877	2,077	84	
1850	2,206	124		1878	1,870	75	81
1851	1,998	113		1879	1,824	72	
1852	2,075	116		1880	2,014	78	
1853	1,812	100	111	1881	2,314	89	
1854	1,937	105		1882	2,435	92	
1855	2,245	122		1883	2,166	81	83
1856	2,125	113		1884	2,145	79	
1857	1,573	83		1885	1,996	73	
1858	2,026	105	97	1886	1,523	55	
1859	1,954	100		1887	1,853	67	
1860	1,665	85		1888	1,635	58	54
1861	1,542	78		1889	1,209	43	
1862	1,523	76		1890	1,375	48	
1863	1,920	94	89	1891	1,263	43	
1864	2,104	102					
1865	1,963	95					

Influence of Season.—Just as it has been seen that erysipelas is apparently more abundant in the colder countries than in the tropics, so it is also decidedly more common, or at least more fatal, in the colder than in the warmer seasons of the year; and this seems to be true for all countries in which careful observations on the point have been made.

As regards London this result is illustrated in the mortality curve given below (Buchan and Mitchell. 'Scottish Meteorological Journal,' 1874-75).

Erysipelas, for all Ages and both Sexes.—(*London Deaths for Thirty Years*, 1845-74).



With respect to the influence of particular kinds of weather on erysipelas mortality, it will be seen by referring to Dr. Longstaff's plate (page 356) that the mortality from erysipelas, like that of scarlet fever, seems broadly to bear an inverse ratio to the rainfall. Cold east winds have often been stated to be conducive to erysipelas.

Influence of Race, Sex, and Age.—It has been alleged that negroes are immune against erysipelas ; but later observations show that such is not the case, though perhaps they suffer less frequently than other races. With them there is no visible rash, but fever, swelling (which pits on pressure), and subsequent desquamation of cuticle. In the brown races the erysipelatous skin assumes a copper colour.

As regards sex, it is usually said that erysipelas is more common among women than men. However it may be with respect to attack, it appears from the table given below that during the ten years 1881–90 the deaths at all ages from erysipelas in England and Wales was greater among males.

TABLE XIV.

Period	Deaths at all ages			Deaths at subjoined ages								
	Male	Female	Total	0–3 Months	3–6 Months	6–12 Months	Total under 1 year	1–	2–	3–	4–	Total under 5 years
Ten years[1] 1881–1890	9,773	8,878	18,651	M. 455 F. 368	136 181	71 102	2,690 2,729	152 174	60 76	30 45	32 35	2,964 3,059
				823	317	173	5,419	326	136	75	67	6,023

Deaths at subjoined ages—*continued*										
5–	10–	15–	20–	25–	35–	45–	55–	65–	75–	85 and upwards
M. 120 F. 97	120 112	149 169	180 201	478 462	824 657	1,105 855	1,383 1,046	1,447 1,199	851 844	152 177
217	232	318	381	940	1,481	1,960	2,429	2,646	1,695	329

The table also gives the age-distribution of the erysipelas deaths during the same years, and it will thus be observed that for the period in question practically 30 per cent. of all the deaths occurred during the first year of life. The deaths under one year were not split up by the Registrar-General into the three sub-periods shown in the table until 1888, but according to the figures for the subsequent years the mortality is very decidedly highest during the first three months of life.

Cause and Mode of Dissemination.—That the ultimate cause of erysipelas is a micro-organism is now sufficiently clear (see p. 93). In many instances this micro-organism unquestionably gains access to the system of the recipient through a wound in the skin, but whether this is always so is uncertain. It has been maintained, indeed, that the microbe of erysipelas is capable of obtaining entrance through the uninjured skin or mucous membranes. The time-honoured distinction between traumatic and idiopathic erysipelas is, of course, based upon the latter supposition. The distinction in question has, however, in recent years received far less acceptance than formerly, and the

[1] All the figures in this table refer to the ten years 1881–90, except those in the age groups 0–3, 3–6 and 6–12 months, which refer only to the years 1888–90.

view has been gaining ground that, even in the case of so-called idiopathic erysipelas, the virus enters the system through some insignificant and usually unobserved breach of surface. In support of this thesis it was pointed out by Trousseau that 'idiopathic' erysipelas may often be traced to a minute breach of continuity in the skin or mucous membrane at the angle of the eye or mouth, or to a slight eczema of the nose; and it is a matter of common experience that this form of erysipelas usually starts from some such point of junction between the skin and mucous membrane, where cracks, fissures, and eczema are peculiarly liable to occur. König succeeded in tracing fifteen out of twenty-nine cases of erysipelas of the head and face to injury of the affected parts, and in many of the remaining cases there was so much swelling at the time of admission to hospital that no satisfactory examination could be made.[1] On the whole, therefore, it seems probable that a wound of some kind, however trivial, is always present.

Erysipelas is certainly infectious, but is less uniformly so than many of the epidemic diseases, such as small-pox, typhus, and measles. The necessity for a wound (if such necessity exists) would doubtless, in part, explain the commonly smaller infectiousness exhibited by the disease among the general public, but even in the surgical wards of hospitals erysipelas often fails to spread. Its prevalence, however, in different hospitals and in the same hospitals at different times has varied largely. To some extent this is very likely due to differences from time to time in the infectiveness of the disease —i.e. to actual variations in the pathogenicity of the streptococcus. An interesting series of cases brought before the Paris Academy, in 1864, by Dr. Blin, and apparently indicating clearly an unusually high degree of infectiveness, are thus recorded by Fagge :—'One of the surgeons at the Lariboisière Hospital had under his care two patients suffering from erysipelas when he was himself seized with it. A medical friend from Guise visited him, and fell ill after returning to that place, where no other case of the disease then existed. That gentleman's servant was attacked, and also a relative who came to see him, and who lived in the neighbourhood. The latter gave erysipelas to his wife, and three members of another family, who were repeatedly in contact with them during their illness, suffered in their turn. From this family the disease spread to two Sisters of Mercy, and they carried it to their home and gave it also to a medical man who attended them; and, lastly, it passed from him to his daughter.'

But other circumstances play an important, though indirect, part in determining the prevalence of erysipelas in particular localities, hospitals, and, as has been often observed, even in particular wards of hospitals. The most important of these circumstances are overcrowding and defective ventilation, filth accumulation, general want of cleanliness, and defective drainage arrangements. In whatever way these circumstance operate, whether by increasing susceptibility, or as affording nidus and pabulum outside the body for the erysipelas microphyte, there can be no doubt as to their operation. '. . . The influence of any epidemic [of erysipelas] is immensely increased by an unhealthy condition of a ward from overcrowding,' says Mr. Erichsen; and this has been the general experience.

An interesting illustration of the effect of filth accumulations in this respect is afforded by the case recorded by Mr. Thomson in the 'Medical Times and Gazette,' December 1856, and also by the late Mr. de Morgan in Holmes' 'System of Surgery.' It appears that the occupants of two beds, one on either side of a window in a large ward on the ground-floor of the

[1] Quoted from Fagge's *Principles and Practice of Medicine.*

Middlesex Hospital, were frequently, during a period of years, attacked by erysipelas. Inquiry elicited the fact that a large uncovered dustbin was situated just below the window in question. Suspicion attaching to this dustbin, it was carefully cleaned out and covered over, erysipelas thereupon ceasing to invade the beds in question. Two years later patients in these beds were again attacked with erysipelas, and examination showed that the dustbin was again in a foul condition and had been left open. It was therefore entirely removed, and the disease disappeared from the ward.

In the year 1874 an epidemic of erysipelas in the Radcliffe Infirmary, at Oxford, was associated by Mr. Netten Radcliffe[1] with a blocking of the drains by fæcal matter and the admission of foul drain-air to the hospital. Dr. König,[2] of Rostock, traced the spread of erysipelas at the hospital there to defective cleansing of the cushions on the operating-table, which were covered with old bloodstains. On discarding these pillows the erysipelas at once ceased.

Among predisposing causes special to the individual, intemperance, want of proper food, and visceral disease are said to be important, It is also often said that liability to erysipelas is increased by a previous attack. Certainly the disease seems to afford little, if any, protection.

Relation to other Diseases.—The question as to what clinical conditions should, on the ground of etiological identity, be included under the term erysipelas is, as has been said, one which it is as yet impossible to answer definitely. Moreover, in the rational study of the origin and natural history of disease, consideration of conditions of causation should take account of possible gradations of genetic relationship between associated maladies, whether or not having similar manifestations. As regards erysipelas, the widely different views which have been, and still are, held upon these matters by different authorities show the subject to be one especially deserving of careful and close study.

And as to this it is here only intended to put forward, provisionally, a few considerations, suggested for the most part by clinical or epidemiological experiences.

With respect, first, to the question of relationship between erysipelas and erythema, it has to be noted that, as often employed, the latter term is one of very uncertain signification. While, on the one hand, there can be little doubt that many so-called cases of erythema are, in reality, mild cases of erysipelas, it is quite certain that there are other conditions of superficial dermatitis which are altogether etiologically distinct.

Next there is the question as to whether erysipelas is 'specific' to the extent of being entirely distinct from various other forms of blood-poisoning which, without inducing any notable spreading dermatitis, give rise to local suppurative adenitis or more remote abscess. Many authorities have answered this question in the affirmative, but the manner in which cases of the latter sort are, in practice, sometimes closely associated with cases apparently of true erysipelas appears to raise great doubt upon the point. In this connection it is important to observe that Fehleisen's erysipelas streptococcus is microscopically and in culture so like the *Streptococcus pyogenes* that some bacteriologists regard the two as identical *quâ* species, and consider the one an attenuated form of the other (see page 94).

But the question under consideration is bound up with that of the relation of erysipelas to certain other diseases, and there are not a few additional epidemiological facts which go to support the thesis of genetic relationship

[1] *British Medical Journal*, 1875, vol. i. p. 651. *Report of Medical Officer, Local Government Board*, 1876, pp. 38–69.

[2] *Arch. der Heilkunde*, 1870, p. 23.

between erysipelas and septic processes as such. It may be mentioned, first, that in this country at least experience has established the interchangeability of *cutaneous* erysipelas and the varieties of the disease characterised by deeper, diffuse, and suppurative inflammation. Clinical observation, too, long since indicated a connection between erysipelas and puerperal fever Attention seems first to have been drawn to this connection during the latter half of the last century, and since that time a large number of facts have been recorded which go to indicate a very close relationship between erysipelas and puerperal fever. Not only has puerperal fever been many times clearly traced to infection carried by medical men to the lying-in chamber from cases of phlegmonous erysipelas in their ordinary practice, but nurses and others suffering from commencing cutaneous erysipelas of the head or face seem without doubt to have set up puerperal fever in 'labour' patients they chanced at the time to be attending. Interesting cases illustrating these points are given by Hirsch, but it must suffice here to say that Hirsch summarises the grounds upon which belief in the etiological connection of the two diseases in question is based as follows :—

'(1) *The coincidence in time and place of the two diseases in epidemic form, both in lying-in institutions and among the population at large.*

'(2) The familiar fact that *women in labour attended by doctors or midwives who were suffering themselves from erysipelas, or had come in contact with erysipelas patients, have taken puerperal fever.*

'(3) The converse fact to (2), that *doctors, midwives, nurses, and other individuals who come into close contact with puerperal fever patients suffer from erysipelas remarkably often; also, that the new-born infants of mothers with puerperal fever die of erysipelas in an unusually large ratio.*

'(4) The fact, vouched for by many observers, that *childbed fever itself has not unfrequently an erysipelatous character*, if I may so speak; or, in other words, that *the disease begins to develop from an erysipelas which mostly arises in the lacerated vaginal mucous membrane.*'

Further evidence of relationship between erysipelas and puerperal fever is found in the similarity of their seasonal incidence, and more strikingly, perhaps, in the closeness with which mortality rises and falls in different years, as shown in the diagram reproduced, by Dr. Longstaff's permission, from 'Studies on Statistics.' In his diagram the rise and fall above and below the mean in the mortality of a number of diseases is indicated for the twenty-six years, 1855–80. With reference to the close resemblance between the curves of erysipelas and puerperal fever, Dr. Longstaff remarked in his original paper, read before the Epidemiological Society in 1880: 'I confess that I find it difficult to avoid the conclusion that they are both due to one poison.' In republishing this paper in his 'Studies on Statistics' in 1890 he has in an appendix given, in another table, similar mortality curves for these two diseases, along with rheumatism, but separately for each of the eleven registration districts of England and Wales, and for the longer period of thirty-four years, 1855–88. In these curves for the different sections of the country the fluctuations are greater than in those for the country as a whole, and, as might be expected, the correspondence between the curves representing the mortality of the three diseases is not always so close as in those based upon the larger figures of England and Wales. Nevertheless, it is conspicuous, and, as Dr. Longstaff remarks, 'far closer than mere chance coincidence would account for;' and he adds, '. . . a relationship which holds good in the several parts of the country, as well as in the entire area, cannot be fortuitous.'

But in addition to the resemblance of the erysipelas and puerperal fever

curves to one another, there is a decided likeness between those curves and that of pyæmia.[1]

In addition, there are broad points of agreement between all the curves in the diagram (representing collectively Dr. Longstaff's 'scarlatinal group' of diseases), in that the elevations and depressions so well-marked in the scarlatina curve are more or less traceable in all the others. There is also a general agreement between the curves in exhibiting a sort of inverse relationship to the rainfall, which is exhibited at the top of the diagram on Sir George Buchanan's method.[2]

One highly interesting result brought out by Dr. Longstaff's inquiries is the close resemblance between the curve of 'rheumatism of the heart' and those of erysipelas and puerperal fever. Is this to be taken as indicating that rheumatism is, after all, a septic disease? The subject is well worth close investigation. In the meantime, it must be remembered that ulcerative endocarditis, the origin of which, according to Fagge, is 'far most frequently in rheumatism,' is apparently a septic process (see page 92).

Lastly, the question arises as to the relationship, if any, of erysipelas to diphtheria. The frequency with which congestion of the fauces accompanies facial erysipelas has long been known. Sir Thomas Watson laid great stress upon soreness of the throat as an early clinical feature, and Mr. Marcus Beck[3] mentions redness and congestion of the fauces as always present in erysipelas of the head. In some cases, known as erysipelatous pharyngitis and laryngitis, the disease is mainly limited to the throat.

In this connection the extensive and very malignant epidemics of 'ery-

[1] The pyæmia deaths were not given by the Registrar-General until 1862, and, as Dr. Longstaff remarks with reference to the pyæmia curve, 'there can be little doubt that the steady rise from that year must be attributed to changes of nomenclature. . . .'

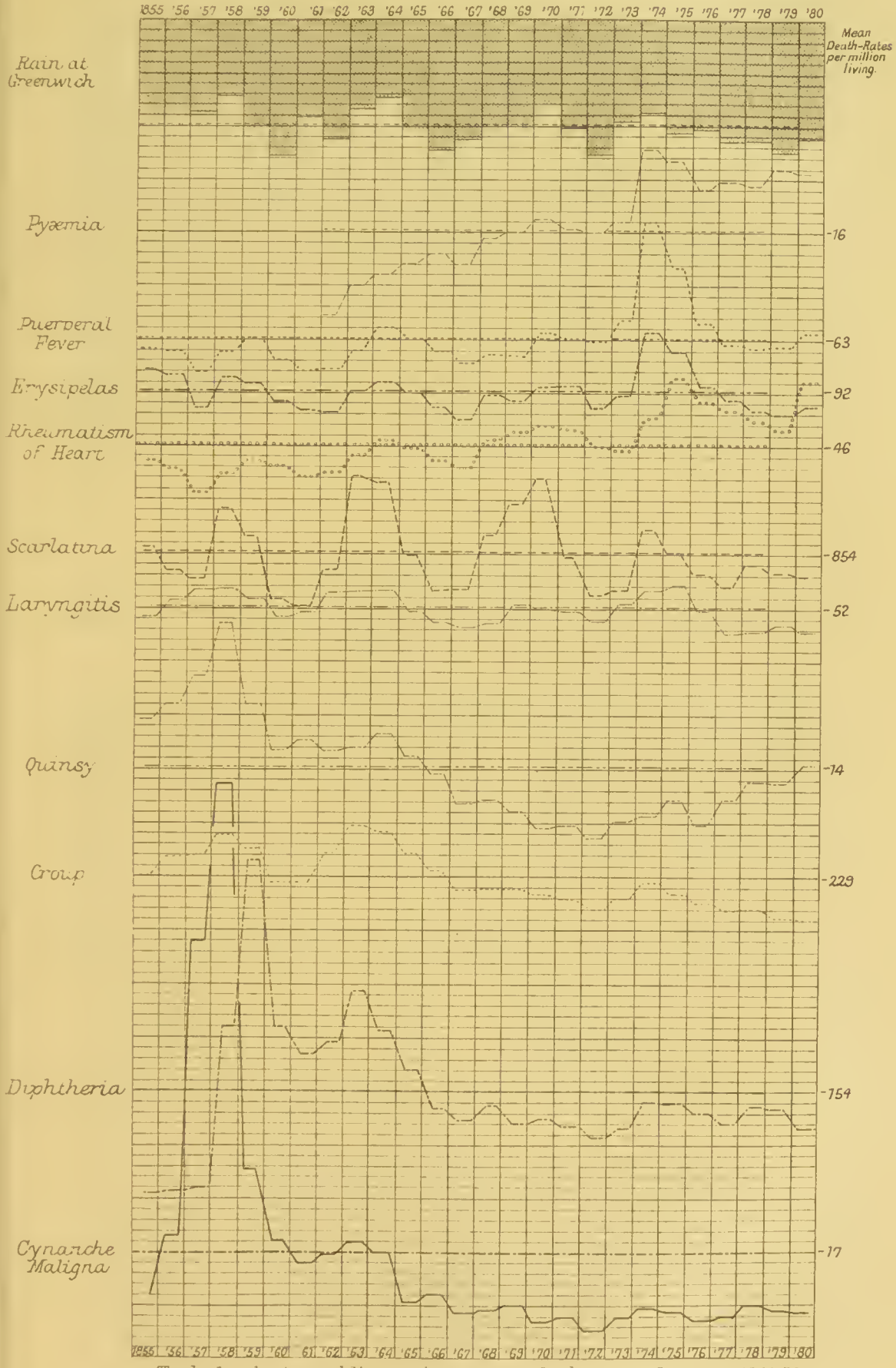
[2] It is only right to add that in the 1890 Appendix, above mentioned, Dr. Longstaff refers to the mortality returns for the further period of ten years, 1879–88, that had become available since the construction of his diagram, which originally embraced the period, 1855–78. In several respects it appears that the resemblances indicated among the curves in that diagram (Plate XLIV.) were not so closely maintained for the later period. The pyæmia curve, indeed, we learn has 'in recent years exhibited no distinct relationship to any of the others.' As regards puerperal fever and erysipelas, the curve for the former disease was above the average for nine of the ten years, and that of the latter was somewhat low, reaching the average only in 1882; but, 'nevertheless a close correspondence in the directions of the fluctuations of the two curves has been maintained.' The scarlet-fever curve has fallen so conspicuously that there is little discernible correspondence between it and other curves. The registered 'croup' mortality has somewhat declined, while that of diphtheria has steadily increased; but as regards their fluctuations these curves have continued to run similar courses; and the laryngitis curve has fairly corresponded with them. Lastly, there has not generally been the same close inverse relationship between these curves and the rain curve as had been previously observed, though in the very wet year of 1879 erysipelas, puerperal fever, and rheumatism of the heart were all at a minimum, while in the dry year of 1884 diphtheria was at a maximum. The discrepancies thus indicated between the results of these two periods were probably in part due to the operation of disturbing causes during the more recent period. Among such disturbing causes may be mentioned certain changes in the classification of deaths which were introduced by the Registrar-General in 1881; improvements in diagnosis and certification by medical practitioners, and increased attention to sanitary matters, which, with better sanitary administration, may have artificially diminished the mortality of some of the diseases in question, and so have tended to obscure natural similarities between them. No doubt also there were other less obvious disturbing causes in operation; but however that may be, the agreements between the curves which were maintained throughout the earlier and comparatively long period of twenty-six years are far too conspicuous to be ignored, and are certainly significant. It will be remembered, too, that as regards erysipelas and puerperal fever, and croup and diphtheria, the fluctuations in the curves have continued to run together.

[3] Quain's *Dictionary of Medicine*.

ENGLAND AND WALES.

Death rates from Erysipelas, Scarlatina & certain other diseases, with rain at Greenwich.

All ages and both sexes.



The broken horizontal lines indicate means for the twenty four years 1855-78.

The curves express the fluctuation per cent above or below the mean. Each division of the vertical scale corresponding to 10%.

The figures at the side give the actual values of the Means of the Death-Rates per million persons estimated to be living.

West, Newman lith.

sipelas' which occurred in North America from about 1841 to 1864 are of especial interest. Throat symptoms constituted an early and conspicuous feature of the disease, and, according to Hirsch, in severe cases the mucous membrane was often found covered with ash-coloured sloughs.

Judging from the accounts of this epidemic, the phenomena of erysipelas and diphtheria seem to have been about equally mixed. Indeed, Volkmann is inclined to regard the disease as 'an affection very closely related to diphtheritis, perhaps even with pure diphtheritis of the throat.' Hirsch, on the other hand, explains that on clinical and etiological grounds he agrees with the American profession in regarding it as malignant erysipelas.[1]

Period of Incubation.—The incubation period of erysipelas would seem to be short, and is given by most clinical observers as usually about four days or less. According to Murchison,[2] it may range from one to eight days, but is usually from one to three or four days. For inoculated erysipelas the incubation period seems to be, as a rule, much shorter. In Fehleisen's inoculations into rabbits with cultures of his erysipelas streptococcus the skin of the ear showed a deep blush after twenty-four hours. In his inoculations in the human subject, with cultures of this organism which were undertaken for the removal of sarcomatous growths, the period varied from fifteen to sixty-one hours.

PUERPERAL FEVER

Synon.: *Childbed Fever; Puerperal Septicæmia.* Fr. *Fièvre puerpérale*; Ger. *Kindbettfieber, Puerperalfieber.*

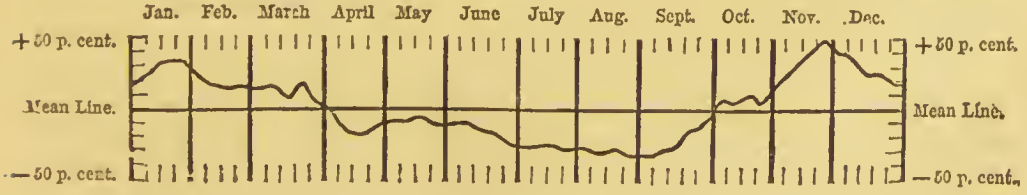
Puerperal fever has occurred in all ages, and seems to have been by no means uncommon in the days of Hippocrates. In more modern times, however, its prevalence was, no doubt, until recently much increased by the establishment and multiplication of maternity institutions. As regards distribution, it occurs in all climates, but, like erysipelas, less frequently in the tropical and sub-tropical than in the higher latitudes. In addition to this apparent preference for the higher latitudes, most observers are agreed that both its prevalence and mortality are greatest in the colder seasons of the year.

With respect to prevalence, Hirsch records that, of 195 epidemics 'for which the time of prevalence is accurately given,' there occurred:—

In winter	66	In autumn and winter	11
In spring	34	In summer	10
In winter and spring	25	In summer and autumn	7
In autumn	21	In spring and summer	5

As to mortality, the incidence upon the autumn and winter months is indicated for London in the following curve.

Puerperal Fever.—London Deaths for Thirty Years, 1845–74. (Buchan and Mitchell.)



[1] *Op. cit.*, vol. ii. p. 398.

[2] *Trans. Clin. Soc.*, vol. xi., London, 1878.

The deaths in England and Wales annually recorded by the Registrar-General as due to puerperal fever are given in the following table. But instead of the rate based upon the proportion of deaths to the general population, which is given in the corresponding tables for other diseases, a rate calculated on the annual number of births has been here substituted. Although this does not take account of still-birth confinements and miscarriages, it seems to be the closest indication obtainable as to the true increase, or the reverse, of mortality from this disease.

TABLE XV.

Year	England and Wales		Puerperal fever deaths to a thousand births	Year	England and Wales		Puerperal fever deaths to a thousand births
	Puerperal fever deaths	Births			Puerperal fever deaths	Births	
1847	784	539,965	1·5	1870	1,492	792,787	1·9
1848	1,365	563,059	2·4	1871	1,464	797,428	1·8
1849	1,165	578,159	2·0	1872	1,400	825,907	1·7
1850	1,113	593,422	1·9	1873	1,740	829,778	2·1
1851	1,009	615,865	1·6	1874	3,108	854,956	3·6
1852	972	624,012	1·6	1875	2,504	850,607	2·9
1853	795	612,391	1·3	1876	1,746	887,968	2·0
1854	954	634,405	1·5	1877	1,444	888,200	1·6
1855	1,079	635,043	1·7	1878	1,415	891,906	1·6
1856	1,067	657,453	1·6	1879	1,464	880,389	1·7
1857	836	663,071	1·3	1880	1,659	881,643	1·9
1858	1,068	655,481	1·6	1881	2,287	883,642	2·6
1859	1,238	689,881	1·8	1882	2,564	889,014	2·9
1860	987	684,048	1·4	1883	2,616	890,722	2·9
1861	886	696,406	1·3	1884	2,468	906,750	2·7
1862	940	712,684	1·3	1885	2,420	894,270	2·7
1863	1,155	727,417	1·6	1886	2,078	903,866	2·3
1864	1,484	740,275	2·0	1887	2,450	886,331	2·8
1865	1,333	748,069	1·8	1888	2,386	879,868	2·7
1866	1,197	753,870	1·5	1889	1,852	885,944	2·1
1867	1,066	768,349	1·4	1890	1,956	869,937	2·2
1868	1,196	786,858	1·5	1891	1,973	914,157	2·2
1869	1,181	773,381	1·5				

From this table it will be seen that puerperal fever attained its maximum recorded mortality during registration times in the year 1874. Nevertheless, it must be noted that, notwithstanding the great advance made in our knowledge of the etiology of this disease and our ascertained ability to control it by cleanliness, ventilation, and especially by antiseptic measures, the last ten years show a higher sustained mortality than any previous decade. This is no doubt largely due to better certification, and is, so far, apparent rather than real, more especially as in 1881 the Registrar-General adopted a system of writing to medical practitioners for further details in all cases returned simply as pyæmia, septicæmia, peritonitis, &c., in women of the child-bearing age—a system which has led to the inclusion under the head of puerperal fever of a number of deaths which would not otherwise have been so classified. If, however, all due allowance were made for improved certification, the period in question would at least not show the decline which we might reasonably have anticipated in the case of this essentially preventable disease.

This is the more remarkable since the application of modern knowledge (as indicated by generally improved hygienic conditions and the use of antiseptics) in the management of lying-in hospitals—institutions which in earlier years were prolific centres of puerperal fever—has led to a very marked decline in the puerperal fever mortality at such hospitals. According to

Dr. Cullingworth,[1] the mortality at the Vienna Lying-in Hospital (the largest in the world) was reduced by hygienic reforms from 28 per 1,000 in the years 1857–62 to 16 per 1,000 in 1863–80, and, subsequently to the introduction of antiseptics, has since fallen to 7 per 1,000. In the similar institution at Dresden the reduction has been from 50 per 1,000 in 1872 to 10 per 1,000 in 1886–87; in the New York hospital, from 60 per 1,000 in 1883 to 2 per 1,000 in 1885–86; and in the Boston hospital, from 55·5 per 1,000 in 1882 and 45·8 per 1,000 in 1883, to 16 per 1,000 in 1884, 6·4 in 1885, and none in 1886. At the Maternité, in Paris, the total death-rate was reduced by sanitary improvements from 93 per 1,000 in 1858–69 to 23 per 1,000, and, after the introduction of antiseptics, to 11 per 1,000. Equally satisfactory progress has been made in our own lying-in hospitals, from which puerperal fever has almost entirely disappeared. From this it seems to follow the sustained mortality from the disease in England must be due to its prevalence outside maternity institutions. Evidently, then, the conditions under which women are confined outside hospitals have not in an equal degree shared in the improvement undergone by the same conditions in hospital practice. Such improvement can only be brought about by a full appreciation of all that is known of the etiology of the disease, and an adequate sense of responsibility on the part of those brought into relation with the lying-in chamber. It must be remembered, as insisted upon by Semmelweiss, that puerperal fever is essentially a septic process, the virus gaining access to the body, in most cases, at all events, through the mucous surfaces of the utero-vaginal passages, to which it may readily be conveyed from case to case by the hands, instruments, and clothing of the attendants, or by sponges, bedding, and other articles. In one or other of the ways indicated puerperal fever has frequently been spread through a series of cases by medical practitioners, midwives, and nurses.

It is, however, not alone from puerperal fever products that the infection may be brought. There is ample evidence to show that it may come from various septic and decomposition sources altogether apart from the lying-in room. Many cases are on record which leave no room for doubt that puerperal fever, as such, has often had its origin in the attendance of women in labour by medical men who had shortly before conducted *post-mortem* examinations, or been in close attendance upon persons suffering from septic maladies.

The ability of erysipelas infection to give rise to puerperal fever has been elsewhere dealt with (p. 355), and there is reason also for thinking that scarlet fever and other infectious diseases may operate in a similar manner; but with regard to such diseases the evidence is not so strong as in the case of erysipelas.

Various other less direct causes play an important part in the origin, or at least the maintenance, of puerperal fever, the most important of which are overcrowding, insufficient ventilation, drainage defects, filth nuisances of all kinds, and want of cleanliness generally. There is clear evidence, for instance, that the past prevalence of the disease in lying-in hospitals, which has already been referred to, was largely due to these causes. It has frequently been recorded that outbreaks in such institutions occurred at times of unusual overcrowding, or were traced to want of ventilation and proper cleanliness; and we have already seen that marked diminution in mortality has followed upon improvement in these respects.

There is no evidence of association between puerperal fever and any particular telluric conditions.

[1] 'Introductory Address at St. Thomas's Hospital in 1888.' Reported in *Public Health*, 1888.

TUBERCULOSIS

Synon.: Fr. *Tuberculisation, Tuberculose*; Ger. *Tuberkelbildung, Tuberculose.*

Tuberculosis—that is, disease which is now known to be causally related to the operation of the so-called bacillus tuberculosis—occurs in the human subject in a variety of clinically different forms, the most notable of which are acute general tuberculosis, tubercular phthisis, tubercular meningitis, tubercular peritonitis, tabes mesenterica, scrofula, and lupus.

But although apparently all due to the same bacillus, the clinical differences between some of these several forms, say, for instance, between acute tuberculosis and lupus, are such as to suggest either corresponding functional differences between the bacilli causing them, or important differences in the tissue-condition of the persons affected. Tubercular disease is by no means limited to the human family. Several of the lower animals, particularly guineapigs and rabbits, as also fowls, have been shown by experiment to be susceptible to it, though in different degrees; and the bovine species suffer naturally, and in large numbers, from a form of tuberculosis known as 'the grapes.' Pigs also not uncommonly suffer from tubercular disease. With reference to the above matters, however, the reader must be referred to the article by Dr. Klein (p. 208, *ante*).

As regards the general history and geographical distribution of tubercular disease, it is certain that in some forms, as, for instance, pulmonary phthisis, it has occurred in all ages, and that it now occurs practically in all countries; but the data for forming even an approximate estimate of the relative prevalence in the past, at different times and places, of this and other forms of the malady are wanting.

In the present day the disease is far more common in some countries than others. Phthisis, for instance, is, taken generally, most prevalent in the countries within the temperate zone, and especially in the more populous parts of such countries. But that no particular temperature *per se* is a bar to its prevalence seems tolerably evident from the facts of its distribution. Thus, although many cold countries, such as Iceland and the Faröe Islands, apparently enjoy a marked relative immunity from phthisis, that malady is very common in North Greenland. On the other hand, as regards the countries with high atmospheric temperature, although phthisis is comparatively rare in Syria, Algiers, and other parts of the North Coast of Africa, it is common in certain parts of India, the islands of the Southern Pacific, and in Mauritius, Réunion, and Madagascar. A notable fact is, that in certain countries, especially Brazil, Australia, and the United States, the disease seems to have decidedly increased of late years.

Mortality.—That tubercular disease occasions an enormous mortality is a matter of common knowledge. It is impossible, however, to ascertain accurately the extent of that mortality, owing to the large element of uncertainty which attaches to the actual cause of many of the deaths, especially among children, ascribed to one or other form of tuberculosis. Hirsch, however, estimates that among civilised communities the average deaths from phthisis amount to one-seventh of the total mortality.

As regards England and Wales, Table XVI., compiled from the Registrar-General's Annual Reports, shows the number of deaths recorded year by year as due to phthisis, and the corresponding death-rates per million of the population. It is gratifying to observe from this table that

TABLE XVI.

PHTHISIS. ENGLAND AND WALES.

Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1838	59,025	3,996		1866	55,714	2,602	
1839	59,559	3,939		1867	55,042	2,539	
1840	59,923	3,897	3,880	1868	51,423	2,336	2,448
1841	59,592	3,822		1869	52,270	2,352	
1842	59,291	3,746		1870	54,231	2,410	
1843	—	—		1871	53,376	2,342	
1844	—	—		1872	52,589	2,271	
1845	—	—		1873	51,355	2,194	2,218
1846	—	—		1874	49,379	2,081	
1847	53,317	—		1875	52,943	2,202	
1848	51,663	—		1876	51,775	2,119	
1849	50,299	—		1877	51,353	2,079	
1850	46,618	2,624		1878	52,856	2,111	2,040
1851	49,166	2,781		1879	51,272	2,021	
1852	50,594	2,826		1880	48,201	1,869	
1853	54,918	3,034	2,851	1881	47,541	1,825	
1854	51,284	2,791		1882	48,715	1,850	
1855	52,290	2,822		1883	50,053	1,880	1,830
1856	48,950	2,601		1884	49,325	1,827	
1857	50,106	2,627		1885	48,175	1,770	
1858	50,442	2,591	2,603	1886	47,872	1,739	
1859	50,149	2,547		1887	44,935	1,615	
1860	51,024	2,564		1888	44,248	1,568	1,635
1861	51,931	2,581		1889	44,738	1,573	
1862	50,962	2,502		1890	48,366	1,682	
1863	51,072	2,476	2,564	1891	46,515	1,599	
1864	53,046	2,541					
1865	53,734	2,541					

during the period in question there has occurred both a relative and absolute diminution in the deaths so recorded. In part this is, no doubt, a matter of nomenclature, but there are independent grounds for thinking that an actual diminution in phthisis mortality has occurred.

Influence of Climate and Season.—Tubercular disease may occur in any climate, and although climatic conditions without doubt exercise a large influence over such malady, it seems probable that this influence has, owing to its having been confused with other factors, been somewhat exaggerated. It has already been pointed out that mere high or low atmospheric temperature is no bar to a considerable prevalence of the disease.

There appears, however, to be a general agreement among observers that, other things being equal, the disease is of a more severe and rapid type in tropical than in temperate climates.

But the fact which seems most clearly indicated by a study of the distribution of phthisis is, that a moist atmosphere with a considerable daily range of temperature is decidedly favourable to its prevalence. How far this is an influence operating directly upon the life-processes of the tubercle bacillus, and how far an influence operating indirectly, by conducing to general susceptibility or to a relaxed and unhealthy condition of the mucous membranes of the recipients, it is difficult to determine.

As regards the influence of season, in this country the phthisis mortality (London) has its annual maximum in March, April, and May, and its minimum in August, September, and October (Buchan and Mitchell).

Influence of Race, Sex, and Age.—No race is exempt from tubercular disease, but the Jews are said to enjoy a relative immunity from it. On the other hand, many coloured races suffer considerably from phthisis, and it has been especially observed that negroes upon changing their natural and primitive habits of life for the conditions associated with civilisation exhibit a very marked susceptibility to it, and this is the more conspicuous if such changed mode of life is accompanied by migration to a temperate climate.

The sex and age distribution of recorded 'phthisis' deaths in England and Wales during the years 1881–90 is shown in Table XVII., compiled from the Registrar-General's Annual Reports.

TABLE XVII.[1]

Period	Deaths at all ages			Deaths at subjoined ages								
	Male	Female	Total	0–3 months	3–6 months	6–12 months	Total under 1 year	1–	2–	3–	4–	Total under 5 years
1881–1890	246,455	227,513	473,968	M. 170	297	556	3,935	2,788	1,357	892	782	9,754
				F. 102	247	528	3,312	2,728	1,318	941	896	9,195
			Totals	272	544	1,084	7,247	5,516	2,675	1,833	1,678	18,949

Deaths at subjoined ages—*continued*										
5–	10–	15–	20–	25–	35–	45–	55–	65–	75	85 and upwards
M. 4,131	5,157	17,595	27,529	59,126	53,952	38,810	21,766	7,578	1,015	42
F. 5,371	10,523	24,877	30,275	59,381	44,226	25,103	12,839	4,907	767	49
9,502	15,680	42,472	57,804	118,507	98,178	63,913	34,605	12,485	1,782	91

It will be observed, as regards sex, that during the period in question, at all ages together, the deaths among males exceeded those among females. This also holds good for the first three years of life, and for the age period 35–45 and upwards. For the intervening periods the deaths among females exceeded those among males. It will be observed that in the age period 10–15 years the recorded mortality among females was double that among males.

As regards age, the deaths registered as due to 'phthisis' fell from the first to the fifth year. After the age period 5–10 they increased up to the age period 25–35, subsequently to which it steadily decreased.

Of the other forms of tubercular disease, it appears, according to Dr. Sims Woodhead's cases, that tubercular meningitis is most common between the third and eighth years of life, but that tubercular disease of the mesenteric glands is commonest at an earlier age, namely in the period from one to two and a half years, which, as will be seen later, may be significant from an etiological point of view.

Cause and Mode of Dissemination.—The discovery of the tubercle bacillus, although a matter of the highest importance and of very great scientific interest, by no means exhausts, or even nearly exhausts, the subject of the etiology of tubercular disease. It has already been pointed out, with respect to microbic diseases generally, that although a given microbe con-

[1] All the figures in this table refer to the ten years 1881–90, except those in the age groups 0–3, 3–6, and 6–12 months, which refer only to the years 1888–90.

stitutes the particulate cause, yet various other conditions are also requisite for the production of the phenomena which we recognise as disease. In the case of tuberculosis this is pre-eminently true, and the 'other conditions' must in practice be regarded as having an importance hardly second to the microbe itself.

It is a matter of common knowledge that tubercular disease is conspicuously prone to occur in different members of the same family, and this also through successive generations. Such might, of course, be regarded—especially in view of the microbic nature of the malady—as a consequence of direct or indirect infection, as ordinarily understood, from case to case; and there can be little doubt but that some instances of multiple attacks in the same family are to be thus accounted for—a matter, however, which will be referred to later. But in view of the great frequency with which this disease is seen to 'run in families' (even though the members of such families are separated, and living under different conditions), and of the slight tendency which it exhibits in general to spread by infection, it seems clear enough that heredity is responsible for a considerable share of its prevalence. Whether this influence of heredity is indirect, depending upon the transmission from parent to child of a special susceptibility, or whether it is direct, involving the actual transmission of the tubercle bacillus in the process of gestation, is a matter which, in the present state of knowledge, cannot be definitely answered; but it is quite possible that it operates now and again in both of the ways suggested. The subject has, however, already been dealt with by Dr. Klein in this volume.

Among other conditions influencing tuberculosis, topographical and telluric circumstances, such as elevation, dampness of soil, hold important place; but for this branch of the subject the reader must be referred to Dr. Copeman's article, vol. i. p. 356 of this work. Certain conditions, however, which belong to the class of hygienic, or, more correctly, unhygienic, conditions remain to be considered here. Foremost among these, as regards that common form of tubercular malady, pulmonary phthisis, is defective ventilation. As evidence of the influence of this factor we have, first, the circumstance that phthisis is most common in large cities, and especially in the most densely populated parts of such cities, where overcrowding and imperfect domestic ventilation are commonly at a maximum, and the free movement of air around dwellings at a minimum. Next we have the more definite and convincing evidence as to the close relationship between defective ventilation and phthisis mortality that is afforded by the past history of life in barracks, ships, prisons, and other institutions. Much important evidence on this point will be found in the Report of the Royal Commission on the Sanitary Condition of the Army which was published in 1858. Up to that time the barracks were overcrowded and insufficiently ventilated, and the phthisis mortality among troops was excessively high. Subsequently to the Report of the Commission the allowance of air-space was increased and the ventilation improved, with the result of a very marked diminution in the phthisis mortality.[1] Similar experience is afforded by the health-history of the Royal Navy, and by that of the occupants of

[1] The overcrowding had been greatest among the Foot Guards, and in that branch of the Service the phthisis mortality had been highest. During the ten years 1837–46 it was 11·9 per 1,000 of strength. For the seven years 1864–70 it had been reduced to 2·3. The mean of the phthisis mortality in the Household Cavalry, Cavalry of the Line, Foot Guards, and Infantry was, for the years 1837–46, 7·89 per 1,000 of strength. In the year 1888 the mortality from phthisis in the British Army at home was 1·2 (See Parkes's *Hygiene*, 8th edit., pp. 582–4).

prisons.[1] The same conclusion is also brought out in another way. In summarising the results of an exhaustive inquiry into the excessive mortality from lung disease in certain districts which had been undertaken for the Privy Council by Dr. Greenhow, Sir John Simon remarked :—'*In proportion as the male and female populations are severally attracted to indoor branches of industry, in such proportion, other things being equal, their respective death-rates by lung disease increased.* And there are medical reasons, which need not now be detailed, for assuming the augmented lung disease to be phthisis.' [2]

But the pernicious effects of defective ventilation which have been so far referred to are due chiefly to the accumulation in the air of the products of respiration (including, doubtless, the tubercle bacillus from phthisical persons) ; the exhalations from the body ; and, to some extent, also to the products of imperfect combustion consequent on defective methods of heating and lighting. Experience, however, has shown that the loading of the atmosphere of mines, factories, and workshops with special kinds of dust produced in different trades is also a potent indirect cause of phthisis, and apparently of true tubercular phthisis.[3] So obvious, indeed, was this result that the expression 'miner's,' 'weaver's,' and 'knife-grinder's' consumption long ago came into popular use. The subject was extensively investigated in the continuation of the inquiry by Dr. Greenhow, to which reference has already been made, and much light has since been thrown upon it by Traube, Zenker, Von Ins, Merkel, Theodore Williams, Watson Cheyne, and others. The dusts in question, it appears, upon being inspired, are taken up by the leucocytes and deposited throughout the lung substance, imparting to the lung in time their particular colour, as, for instance, in the case of the black lung of the miner and the red lung of the oxide-of-iron worker. But their ability for harm, *quâ* phthisis, appears to depend largely upon their hardness and angularity. It has long been known that, of the various dusty trades, those of the steel and stone workers are associated with particularly high mortality from lung disease. Coal-miners have, it is true, suffered heavily from phthisis, but it now seems probable that where this has occurred it has rather been due to the general effects of defective ventilation of the mines than to the inhalation of coal dust. When coal-mines are well ventilated, the phthisis mortality among the miners does not appear to be excessive, though the 'carbonisation' of their lungs still occurs. It has, indeed, been held by some observers (as Seltmann and Merkel) that coal dust *per se*, so far from conducing to phthisis, affords some protection against it. On the whole, it would seem that, as a factor in the causation of phthisis, inhaled dust operates in an entirely indirect manner, its operation depending upon its ability to give rise to a

[1] For evidence as to the high mortality from phthisis at Millbank Prison in former times, *see* Dr. Baly's paper, *Medico-Chir. Trans.*, 1845, xxviii. 113.

As regards foreign prisons, an interesting case is quoted by Parkes. In the defectively ventilated prison of Leopoldstadt, at Vienna, during the years 1834–47 there died of phthisis 51·4 per 1,000 of the prisoners, while in the well-ventilated House of Correction in the same city during the years 1850–54 only 7·9 per 1,000 died. The comparative length of sentences in the two cases was not given, but Parkes considered that 'no correction on this ground, if needed, could account for this discrepancy (Parkes's *Practical Hygiene*, 8th edit., p. 168).

[2] *Third Report of the Medical Officer of the Privy Council*, 1860, p. 30.

[3] Space does not admit of a review of the evidence as to the relation to one another and to tubercular phthisis of the lung ailments associated with different trade processes. The subject is discussed by Pye Smith in Fagge's *Principles and Practice of Medicine*, 2nd edit., vol. ii. p. 239. It is sufficient here to say that the tubercle bacillus has been found in knife-grinder's and potter's phthisis.

catarrhal or mechanically injured condition of the mucous lining of the lungs, and so to favour the entrance and activity of the tubercle bacillus.

The relation of food to the propagation of tuberculosis is a subject of great importance. Deficient and defective food no doubt indirectly predispose to tubercular infection, by leading to impaired vitality. But it is as a possible carrier of the tubercle bacillus that food, in this connection, assumes its special importance. It has already been seen that cattle suffer in considerable numbers from tubercular malady, and although the identity of such malady with human tuberculosis may not be completely established, it is in a very high degree probable that the two are but different manifestations of an identical cause. If such is the case, the danger to man as regards cattle is a double one, for infection may doubtless occur both by the ingestion of milk and flesh. The frequency with which the young children of healthy parents suffer from tabes mesenterica is, as Dr. Sims Woodhead has pointed out, very strongly suggestive of infection by milk.

As regards meat, there are good grounds for believing that infection may result in man from the consumption of the imperfectly cooked flesh of animals that have died of tubercular disease, and the important question has arisen as to whether the whole of the flesh of such animals is unfit for human food, or only those parts in which tubercular lesions are manifest. The evidence at present afforded by laboratory experiments on this subject is somewhat conflicting, and the question cannot be said at present to have been definitely solved. The whole matter is now the subject of inquiry by a Royal Commission. Possibly the infectivity of the tissues generally of tuberculous animals, in contradistinction to that of those tissues which are the seat of evident tubercular changes, may depend upon the activity or otherwise of the tubercular ailment at the time of the animal's death. But further evidence on the matter is wanted.

Lastly, reference must be made to the so-called contagiousness of phthisis. This question has been forced to the front during recent years by Koch's demonstration of the microbic character of the malady; and there can hardly be any doubt that phthisis may be spread by infection from case to case, a view which has long been generally held in Italy, Spain, and Portugal, and was advocated by Galen, Morgagni, Dr. Wm. Budd, and others.

Cases in which the disease seems almost certainly to have been transmitted from husbands to wives have been recorded by Dr. Weber[1] and other observers, and the rapidity with which cases of acute phthisis are in practice occasionally seen to succeed one another in members of the same family, but of varying ages, are highly suggestive of infection. On the other hand, the extreme rarity with which the nurses in consumption hospitals are attacked, even though they are exposed to more or less continuous opportunities of infection, seems to indicate that the infective power of the disease is of a very low order, and it has, indeed, been held by some altogether to negative the idea of its infectiousness. The reconciliation of the apparently conflicting evidence is probably to be found in the vast importance, as regards tubercular disease, of the indirect causes, which has been insisted upon above. It is not enough that the tubercle bacillus should be taken into the lungs or alimentary canal—it must also there meet with a favourable soil if it is to give rise to tubercular disease. No amount of negative evidence, therefore, is valid as an objection to the theory of infectiousness. A broad argument for the infectiveness of phthisis is probably to be found in the fact of the increase of the disease during recent years in certain countries, such as Australia and the United States, which have been subject to considerable

[1] *Clin. Soc. Trans.* 1874.

immigration from infected countries, and, in the former case, to considerable immigration of persons affected with the disease. This is quite consistent with a theory of the importation of the disease, and its subsequent spread by infection.

Relation to other Diseases.—It has often been maintained that malarial disease is antagonistic to tuberculosis, but there seems now a considerable doubt on this point. It appears to be a fact, however, that phthisis is seldom seen in the subjects of gout, and, more curiously, it is true that mitral stenosis is almost never found in persons who die of phthisis.

CROUP

Synon.: *Cynanche Trachealis, Cynanche Stridula.* Fr. *Le Croup*; Ger. *Häutige Bräune, Der Croup*; It. *Laringitide Membranacea.*

The doctrine of croup as a disease *sui generis* seems to have been first especially insisted upon by Dr. Home, of Edinburgh (1765), in his 'Inquiry into the Nature, Causes, and Cure of the Croup.' In 1801, Dr. John Cheyne followed upon the same lines. But cases of what Hirsch considers must have been croup had been described as early as 1576 by Baillon in Paris. During the seventeenth and eighteenth centuries further cases, also regarded by Hirsch as croup, were referred to as 'catarrhus suffocativus' by different observers.[1] In 1821, however, Bretonneau maintained the identity of membranous croup and diphtheria, and thus initiated a controversy which has continued to the present day.

It is only proposed here to refer to a few points which must be taken into consideration in attempting to arrive at a conclusion as to the nature of 'croup,' and its relation to diphtheria.

It was admitted by Bretonneau, and has since been abundantly proved, that an inflammation of the laryngeal mucous membrane set up by irritants such as boiling water may result in an exudation of lymph and the formation of a false membrane indistinguishable to the eye from a diphtheritic membrane. There is, therefore, no *à priori* ground for asserting that a membranous laryngitis must of necessity be a diphtheritic process. Further, there would seem no reason to doubt that an inflammatory condition of the mucous lining of the larynx and trachea may be, and sometimes is, set up by non-diphtheritic causes, among which cold and damp doubtless play an important part, even if bacteriology should ultimately show it to be but an indirect part.

When, however, we come to consider the title of 'croup' to be regarded as a separate and specific disease, or when we consider the alleged identity of croup and diphtheria, other important considerations must be taken into account.

Table XVIII. shows the mortality recorded as due to 'croup' year by year since the commencement of registration.

On reference to the table, it will at once be observed that of late years the number of deaths returned as croup has undergone a decided diminution, both absolutely and relatively to the population. Now this is not the case with the diseases of the respiratory system, in the causation of which cold

[1] Among them, Blair, of London, in his *Observations in the Practice of Physic*, 1718.

and damp unquestionably play an important part; and it would seem, therefore, that the deaths ascribed to croup are by no means all of an ordinary catarrhal character.

TABLE XVIII.

Showing Year by Year the Deaths in England and Wales certified as Due to 'Croup,' and the corresponding Death-rates per Million Living.

England							
Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period	Year	Total deaths	Death-rate per million living	Average annual death-rate per million living for each quinquennial period
1838	4,463	302	270[1]	1866	5,168	241	208
1839	4,192	227		1867	4,387	202	
1840	4,336	282		1868	4,491	204	
1841	4,177	268		1869	4,478	202	
1842	4,457	—		1870	4,302	191	
1843	—	—		1871	4,116	181	184
1844	—	—		1872	3,640	157	
1845	—	—		1873	4,282	183	
1846	—	—		1874	5,010	211	
1847	3,338	—		1875	4,542	189	
1848	3,777	—		1876	4,204	172	154
1849	4,038	—		1877	3,910	158	
1850	4,322	243		1878	4,061	162	
1851	4,180	236	224	1879	3,574	141	
1852	4,058	227		1880	3,571	138	
1853	3,660	202		1881	3,594	138	163
1854	3,998	218		1882	4,609	175	
1855	4,419	239		1883	4,591	172	
1856	5,207	277	276	1884	4,748	176	
1857	5,279	277		1885	4,235	156	
1858	6,220	319		1886	3,685	134	126
1859	5,636	286		1887	3,979	143	
1860	4,380	220		1888	3,632	129	
1861	4,397	219	288	1889	3,241	114	
1862	5,667	278		1890	3,145	109	
1863	6,957	337		1891	2,638	91	
1864	6,777	324					
1865	5,921	280					

On the assumption that 'croup' is a separate specific disease, the diminished mortality ascribed to it might, of course, be referred to sanitary improvements, or to a natural decline in the malady. Nevertheless, there are grounds for believing it to be due to improved diagnosis; for thinking, in fact, that some of the deaths which in former years would have been returned as 'croup' are now correctly returned as diphtheria.

For that many cases of diphtheria have been, and still are, ascribed to croup is well known. Inquiry very frequently shows that antecedent to, or concurrently with, an increase in the deaths from diphtheria there has also been an increase in the deaths returned as 'croup;' and it also brings to light instances of diphtheritic paralysis following upon attacks of so-called 'croup.' Moreover, Dr. Longstaff's curves, exhibiting the variations year by year (1855–80) in the mortality from different diseases, among them croup and diphtheria, show that 'since 1861 the curves of croup and of diphtheria have been so very similar as to suggest that, at the very least, there is great confusion in the diagnosis of the two diseases, and even to be a weighty argument in favour of those pathologists who maintain their identity.'[2]

[1] For four years only.

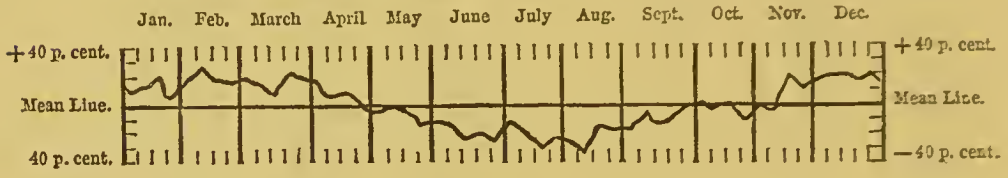
[2] *Studies in Statistics*, p. 316.

The facts thus indicated seem to make it clear that many of the 'croup' deaths are really due to diphtheria.

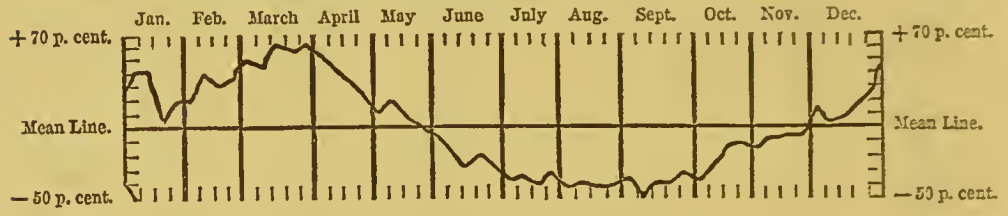
Here, again, however, it must not be concluded that the whole problem is solved. Dr. B. A. Whitelegge has pointed out that the seasonal curve of croup approximates more closely to the laryngitis curve than to that of diphtheria (*see* curves below); that 'croup is more fatal to males than females upon the whole, the mortality among females being lower up to fifteen years of age. The highest mortality in both sexes occurs in the second year of life. In all these respects "croup" is allied to laryngitis rather than diphtheria.'[1]

SEASONAL MORTALITY[2]

Croup, for all Ages and both Sexes.—London Deaths for Thirty Years, 1845–74.



Laryngitis, for all Ages and both Sexes.—London Deaths for Thirty Years, 1845–74.



The provisional conclusions, then, to which the above considerations seem to point are:—

1. That a considerable number of cases of so-called 'croup' are, without doubt, cases of diphtheria.

2. That this, however, is not true of all such cases.

3. But that those which remain, not being of diphtheritic origin, are probably cases of laryngitis, or tracheitis, of a kind more closely allied etiologically to bronchitis.

PNEUMONIA

Synon.: *Inflammation of the Lungs, Peripneumony.* Fr. *Pneumonie*; Ger. *Lungenentzündung*; Ital. *Pneumonitide.*

In considering pneumonia from the point of view of epidemic disease we are, for the present day, mainly concerned with the form known as acute lobar pneumonia, occurring as a so-called idiopathic affection. It must be premised, however, that this restriction is provisional only, for, as will be seen

[1] *Hygiene and Public Health*, by B. A. Whitelegge, M.D., B.Sc., D.P.H., 2nd edit., p. 301.

[2] These curves are reproduced, by permission of Dr. Buchan and Sir Arthur Mitchell, from their papers already referred to.

later on, it is not improbable that, in dealing with the subject from this narrowed view-point, we may be including under a common name two or more maladies which, though superficially identical, are due to different causes; while, at the same time, we may be separating from one another, on the ground of clinical or apparent etiological difference, manifestations due to one and the same cause.

Acute lobar pneumonia was for a long time regarded as a non-specific inflammation, dependent usually upon exposure to cold and damp, the fever and general symptoms being considered as secondary to the local inflammatory action. Of late years, however, this view has largely lost ground, and it has become more and more recognised that in its sudden onset, its rapidly attained pyrexia, the definite course of its symptoms, its relatively constant duration, and its sudden defervescence, pneumonia rather exhibits the characters of a specific fever, with inflammation of the lung as its most prominent local manifestation. This latter thesis, moreover, as contrasted with the older one, derives some indirect support from the fact that pneumonia in its distribution in space, as well as in its relation to season and certain particular kinds of weather, exhibits some points of contrast to bronchitis—a disease also, and upon stronger evidence, held to depend upon cold and damp. Then, again, the existence of a specific [1] fever characterised by pneumonia, or, perhaps, of several kinds of such fever, is rendered probable by the frequency with which pneumonia has been observed to occur in epidemics, and by the fact that it is certainly at times infectious. Lastly, bacteriology is leading to the same general conclusion, and bids fair to put the doctrine of pneumonia upon a more satisfactory basis.[2]

Pneumonia may be traced in the earliest medical writings, though it is only since the introduction of the stethoscope that a definite distinction has been drawn between it and other diseases of the chest. It seems likely that in former times pneumonia was more prone to occur in epidemic form than is the case in the present day. At all events, there are not a few records of its wide prevalence in the sixteenth, seventeenth, and eighteenth centuries in Italy, Germany, France, and especially in Switzerland, where it has long been known as 'Alpenstich.' The earliest records of epidemic pneumonia in England and North America seem to date from the eighteenth century.

During the present century an extensive pandemic diffusion of pneumonia occurred in North America from the years 1812 to 1825, as well as various other epidemics in the same country, both during the earlier and more recent years of the century. In 1853–55 pneumonia was epidemic at Julianchab, in South Greenland. As regards Europe, a number of epidemics have, during the present century, been recorded for Italy, France, Germany, Switzerland, and Great Britain, a few in Norway and Denmark, and one in Iceland (1863). Speaking generally, however, the European epidemics of the present century, as compared with those of previous centuries, have, according to Hirsch, been mostly far apart and confined within narrow limits.[3] Two epidemics are recorded for India (Punjab), in 1875 and 1882. The most recent epidemics of importance in this country occurred at Middlesbrough in 1888, and at Scotter (Lincolnshire) in 1890.[4]

[1] The word *specific* is, of course, used here only in the relative sense which is consistent with the views expressed in the introductory section.

[2] For the bacteriological evidence so far available see p. 123, *ante*.

[3] For fuller information as regards epidemic pneumonia see Hirsch, *ibid.* vol. iii. p. 125.

[4] See Reports by Drs. Ballard and Parsons respectively, in the *Annual Reports of the Medical Officer to the Local Government Board for* 1888 *and* 1890.

Pneumonia, like bronchitis, occurs more or less extensively over nearly the whole world, though certain localities, notably Lower Egypt and Tunis, seem to enjoy a marked relative immunity from it. As already mentioned, however, there is not the close correspondence between the distribution of pneumonia and bronchitis which would be expected upon the hypothesis of their being due to one and the same cause, such as exposure to cold and damp. Thus, the prevalence of bronchitis appears to increase towards the northern latitudes—Iceland, the Faröe Islands, Sweden and Norway, being among its principal seats. This is not the case with pneumonia, which does not seem to be particularly prevalent in the countries named; occurring there, indeed, according to Hirsch, only to a moderate extent. Particular countries in other latitudes might also be cited in which the distribution of these diseases does not closely correspond. As regards England, it has been pointed out by Dr. Longstaff[1] (for the period 1876–80) that pneumonia was comparatively much more fatal than bronchitis in some registration counties, whereas in other registration counties the opposite was the case.

Nevertheless, there can be no doubt that the amount of both pneumonia and bronchitis is largely influenced by cold, but such influence *is greater in the case of bronchitis than in that of pneumonia*. This is brought out, among other ways, by a study of the seasonal mortality of the two diseases. As regards both, the London weekly deaths are above the mean from November to April, and below it from May to October. The pneumonia deaths, however, rise above the mean about a week earlier (last week in October), and fall below it about a fortnight later (first week in May), than the bronchitis deaths. The absolute maximum of pneumonia deaths is in December, that of bronchitis deaths in January. The absolute minimum of both is in August. But the rise and fall in the deaths from both these diseases, above and below the mean, which correspond broadly in point of time to the colder and warmer seasons of the year, are more conspicuous in the case of bronchitis than in that of pneumonia.

But in addition to the general agreement (though subject to differences of detail) exhibited by the seasonal, or week-to-week, mortality of bronchitis and pneumonia, there is a decided general agreement between the extent of mortality of these diseases in different years, and this also appears to have a relation to temperature. Dr. Longstaff, in the paper to which reference has just been made, has compared the curves indicating the mortality of these diseases from year to year (London deaths, 1850–82) with one another, and also with a curve representing the number of cold days at Greenwich in each year. The result obtained is that the year-to-year mortality curves of bronchitis and pneumonia correspond closely in direction and both bear a general resemblance to the 'cold-day' curve; but here again, as in the seasonal curves, the fluctuations are greater in the bronchitis curve than in that of pneumonia.

The general result of the considerations above put forward, independently of the results of bacteriological research, is that, although cold is doubtless an important indirect or predisposing cause of pneumonia, it is not the ultimate cause.

Further analytical study of the influence exerted upon bronchitis and pneumonia mortality by cold shows that this influence is dependent, not alone upon the mere degree of coldness of the atmosphere, but largely also upon sudden and frequent fluctuations of temperature. This seems to be brought out clearly by the observations of many observers, such as Huss for

[1] 'Phthisis, Bronchitis, and Pneumonia,' by G. B. Longstaff, M.A., M.D. (*Epidem. Soc. Trans.*, 1883, vol. ii. N.S.). Also reprinted in *Studies in Statistics*.

Sweden, Eschbaum for Bonn, Schramm, and also Klinger, for Bavaria, Chanseaux for Paris, and Gordon for India. Several Continental observers have ascribed an important influence in particular epidemics to relative absence of rain and lowness of the subsoil water. At Middlesbrough, Dr. Ballard noted that the epidemic made progress during the weeks of drought, but was apparently held in check by abundant rain.

It has already been suggested that the cases commonly grouped together under the head of lobar pneumonia may be instances of two or more different diseases. Bacteriological evidence, so far, is consistent with this view. In the Middlesbrough cases, for instance, Dr. Klein failed to identify either the micrococcus of Friedländer or the diplococcus of Fränkel and Weichselbaum, but he found a short and distinctive bacillus, which apparently stood in causal relation to the malady. For epidemiological suggestions on the point we have the fact of the differences in type of pneumonia from time to time, and the differences in its infectiousness. As regards type, it is to be noted that pneumonia occurring in epidemic form has very commonly exhibited a typhoid character, with somewhat prominent gastric and intestinal symptoms, and a high mortality. This might, perhaps, indicate a class distinction between the pneumonia of such epidemics and that which occurs sporadically. Again, suggestion of the spread of the disease by direct infection is frequently met with in the literature of pneumonia, and there are particular cases on record which seem to show undoubtedly that the malady does at times spread by infection. Of such particular instances may be mentioned some cases which occurred in 1876, and were reported by Dr. Patchett in the 'Lancet' for 1882, and a series of cases in 1879, reported by Dr. F. H. Daly in the 'Lancet' for November 12, 1881. Also cases recorded by Mr. Wynter Blyth in his 'Manual of Public Health,' page 415, and by Dr. Ballard in his Middlesbrough report.

The variations, then, in the property of infectiousness apparently attaching to pneumonia at different times and places are, like the variations in type, consistent with the view of there being different kinds of pneumonia. Differences in the age incidence, which will be referred to later, may also possibly bear a like interpretation, and the subject is well deserving of very careful inquiry. It must, however, be remembered in such inquiry that variations in type, infectiousness, and age incidence, do not necessarily imply specific distinctions in the ailments exhibiting them. They may all be thought of as possibly representing differences in the virulence of one and the same contagium vivum. That this may be so as regards differences of type is matter of common knowledge; and there are reasons for thinking that it may likewise be so as regards infectiousness and age incidence. It is possible therefore that ordinary sporadic lobar pneumonia is capable, under conditions especially favourable to the pathogenicity of its contagium vivum, of passing from time to time into an epidemic form of a typhoid character, with a high fatality and an appreciable degree of infectiousness.

No race is exempt from lobar pneumonia, but many coloured races, particularly negroes, are especially susceptible to it. As regards sex, the mortality at all ages together, according to Dr. Longstaff, is greater for males than females, almost in the proportion of three to two. The disparity is most marked at ages 35–65, when males suffer more than females, in the proportion of two to one. It must not, however, be concluded that, case for case, pneumonia is more *fatal* to males than females. As a general rule the reverse is the fact.[1] According to Huss's statistics, the relative

[1] See article on 'Pneumonia,' by the late Dr. Wilson Fox, in Reynolds's *System of Medicine*, 1st edit. p. 689.

fatality among the two sexes, given an equal number of attacks for each, is males ten, females fourteen. It would thus appear that more males die of pneumonia than females because many more are attacked—that, in other words, the liability to attack is much greater in males, but the liability to death if attacked is greater in females.

With respect to age, the recorded mortality is highest at the extremes of life, being, however, about three times as great in the first year of life as in old age. It falls very rapidly through the first three years, and more gradually to the thirteenth year, when it attains its minimum. Subsequently it rises steadily throughout the remainder of life. The fatality, or case mortality, in relation to age, differs from the mortality in that it is greatest in advanced life. Except, perhaps, for the first year or two of life, it is low during childhood and adolescence, but after about the thirtieth year it increases steadily with advancing years.

Of the general fatality of pneumonia at all ages together it is difficult to speak definitely, since this varies considerably in different times and places. Among Huss's cases (2,618 in number) it was 10 per cent. of the attacks. By other observers it has been found to range considerably above and below this.

The Middlesbrough epidemic pneumonia differed from that of ordinary experience, among other things, in having an especially high fatality (21 per cent.); in that its fatality at all ages together was greater among males than females; and that its incidence upon age, as judged by the number of deaths, in relation to the numbers living at each age period, was greater upon the middle and higher ages than is usual.

Among predisposing causes of pneumonia, other than those which have been referred to, must be mentioned unwholesome conditions of life generally. The records of pneumonia epidemics seem to leave no doubt that overcrowding, defective ventilation, emanations from sewers, filth accumulations, and the like, have had important influence upon the prevalence and type of the disease. As evidence of this we have, as Hirsch says, 'not only the epidemic outbreak and prevalence of pneumonia in confined and circumscribed buildings—barracks, prisons, and such-like—where the factor in question has been peculiarly noticeable at the time of the outbreak and so long as it lasted, but also the fact that in a number of epidemics which have spread over whole villages, those streets or houses have suffered most that were principally exposed to the particular harmful influences.'

In the epidemic at Middlesbrough, Dr. Ballard found good reason for attributing considerable influence to defective drainage as an agency in the incidence of the disease.

As regards influences special to the individual, fatigue and debility predispose both to attack and death. Persons whose constitutions are damaged by the abuse of alcohol are well known to be particularly liable to die of pneumonia.

The not uncommon occurrence of pneumonia in association with other diseases, notably enteric fever, malaria, and influenza, raises the question as to the relationship of the pneumonia in such cases to the diseases in question. Is it, for example, that in certain instances, and owing to some relatively unusual conditions, the enteric fever virus, instead of selecting the intestines for its chief local operations, attacks also, and perhaps most conspicuously, the lungs? Or, again, is it that the diseases in question predispose to independent attack by pneumonia? As a third conceivable hypothesis, it might be suggested that the cases under consideration are examples of hybridism. It is impossible at present to find definite answers to these questions. Perhaps the phenomena referred to may in some cases be due to one, and in some to another, of the causes suggested.

A question allied to those above raised presents itself in connection with the particular outbreaks of pneumonia in Middlesbrough and Scotter. They were apparently outbreaks of one and the same disease, and a disease, moreover, which in certain particulars differed from pneumonia as ordinarily met with. Now the fact, speaking broadly, that these unusual epidemics of pneumonia had some sort of time relation to the pandemic diffusion of influenza throughout the world might suggest a relationship between the two diseases—that the pneumonia of Middlesbrough and Scotter was, in fact, an expression of influenza. Against this view it might be urged that the pneumonia at Middlesbrough was epidemic more than a year before the European prevalence of influenza occurred; but with respect to this there is the fact that, as early as February 1887 an epidemic of an unknown infectious disease had occurred at Northallerton, in Yorkshire, and had been considered by the late Dr. Page, who investigated it for the Local Government Board, as allied to, if not actually identical with, epidemic influenza. Another objection to the thesis, however, is found in the fact that the pneumonia which occurred at Middlesbrough in 1888, although unusually abundant in quantity, seems in some respects, at least, to have resembled in character the pneumonia met with in that particular locality in previous years. On the other hand, it must be noted that in the incidence of its mortality upon age the Middlesbrough pneumonia exhibited a greater resemblance to influenza than to the usual pneumonia of this country; and lastly, it is somewhat suggestive to note that in two out of five cases of croupous pneumonia *following influenza*, Dr. Klein[1] has found 'in large numbers, almost in pure culture,' the particular micro-organism which he identified as the cause of the Middlesbrough pneumonia.

YELLOW FEVER

Synon.: Fr. *Fièvre jaune*; Ger. *Gelbes Fieber*; It. *Febbre gialla.*

History and Geographical Distribution.—Yellow fever is endemic only in certain comparatively limited areas of the earth's surface, whence it from time to time extends in epidemic and even pandemic fashion—thus, as in many other respects (though on a different scale), strikingly resembling cholera. Its two principal endemic centres are: (1) The coast of the Gulf of Mexico and the West India Islands; and (2), a limited portion of the West Coast of Africa, notably Sierra Leone. Whether the disease originated independently at these two centres, or whether, as seems more probable, both *à priori* and in view of the recorded behaviour of the disease in more modern times, at one only of them, being subsequently carried to the other, it is impossible to decide definitely from the historical data available. The difficulties of an inquiry into the point are, moreover, increased by the fact that the severe forms of malarial sickness have very frequently been mistaken for yellow fever. And although there is suggestion that yellow fever was prevalent among the natives on the shores of the Gulf of Mexico prior to the discovery of America, it is impossible to affirm this with confidence. But on the whole, having in view the fact that the first *reliable* accounts of the disease in Africa are of a considerably later date than those in the West

[1] Page 128, *ante*.

Indies, it seems likely, as Hirsch says, that the West Coast of Africa 'was infected from the Antilles, afterwards becoming an endemic focus when the disease got naturalised,' and that in the West Indies we have the head-quarters and original home of the disease. Nevertheless, yellow fever apparently existed on African soil before the first African epidemic of which we have record, since this epidemic, which occurred at St. Louis (Senegal), is said to have been traced to importation from Sierra Leone.

In any case, the first unequivocal accounts of yellow fever in any country come from the West Indies towards the middle of the seventeenth century, when the disease was very fatal in Guadeloupe, appearing about the same time at Barbadoes and Cuba. The first known epidemic at Vera Cruz, the principal seat of the disease on the Mexican coast, seems to have occurred in the year 1699. On the West Coast of Africa, the first recorded outbreak was that already referred to at St. Louis, and occurred in 1778. Subsequently to the dates given the disease has been very frequently prevalent on the Mexican coast and the West India Islands, and on the West Coast of Africa. The particular localities in those endemic centres at which the most frequent prevalences have occurred are as follow:—*Mexican Coast*—Vera Cruz and Tampico; *West Indies*—Martinique, San Domingo, Jamaica, Guadeloupe, Antigua, Cuba, Santa Cruz, and St. Thomas; *West Coast of Africa*—Sierra Leone and Senegambia. During and since the eighteenth century yellow fever has also frequently extended up the east coast of North America, the highest points reached being apparently Halifax (44° 39′ North) and Quebec (46° 50′ North). It first appeared in New York in 1693, and has since been more or less prevalent there on some twenty-one occasions, the last of which was in 1870.

The places in the United States, in addition to New York, which have suffered the most frequent visitations are New Orleans, Philadelphia, Charleston, Mobile, Pensacola, Norfolk, and Baltimore. In New Orleans, judging from the mortality table given by Dr. Sternberg,[1] the disease was almost continuously present from 1817 to 1858. But with respect to yellow fever in the United States Dr. Sternberg remarks that, 'although it has occasionally prevailed as an epidemic in every one of our seaport cities as far north as Boston, and in the Mississippi Valley as far north as St. Louis, it has never established itself as an epidemic [? endemic] disease within the limits of the United States.'

The most important epidemics in the United States were the following:—1793, in Philadelphia (deaths 4,040, said to have equalled 10 per cent. of the population); 1797, in Philadelphia (deaths 1,300); 1798, Philadelphia and other places, including New York (deaths in Philadelphia, 3,645, said to have been 80 per cent. of attacks; in New York, 2,080); 1853, States of Florida, Alabama, Louisiana, Mississippi, Arkansas, and Texas (deaths in New Orleans, Louisiana, 7,970); 1867, States of Texas and Louisiana (deaths in New Orleans, 3,093; in Galveston, Texas, 1,150); 1873, States of Florida, Alabama, Mississippi, Louisiana, and Texas; 1878, 'the last and most extended epidemic' in the United States (Sternberg), invaded 132 towns, chiefly in Louisiana, Tennessee, Alabama, and Mississippi (deaths 15,934, attacks 74,000). Several of these outbreaks were clearly traced to importation from one or other of the endemic centres of yellow fever, and there can be little doubt that they were, in fact, all due to this cause.

As regards South America, Hirsch considers that prior to the year 1850 there are only records of two undoubted epidemics, both of which occurred

[1] *Report on the Etiology and Prevention of Yellow Fever*, by George M. Sternberg, Lieut.-Col. and Surgeon, U.S. Army, p. 44.

at Guayaquil, the first in 1740, and the second in 1842, and both of which are said to have been due to importation. In October 1849, however, yellow fever was imported into Bahia, and soon spread to Rio Janeiro, Pernambuco, and other places. Since that time Brazil has, apparently, seldom been free from the disease, and the question arises whether it has not become truly endemic there. In 1854 the disease was carried to Peru, and in the next few years it spread to many towns on the coast. Chili has so far been exempt (Hirsch).

In addition to its distribution in America and Africa, yellow fever has in not a few instances been imported into Europe, but except in the Iberian peninsula it has never attained any large epidemic proportions. During the several epidemics which occurred in Spain in the eighteenth century, the disease was for the most part limited to Cadiz, but in the years 1800–1804, when it again prevailed in Cadiz, it spread largely over Andalusia, extending to the seaboard of Murcia, Valencia, and Catalonia. In 1810 it broke out once more in Cadiz, Cartagena, and Gibraltar, and 'in the two following years it appeared at the same places anew, and from them it again spread through several of the coast towns of Granada, Murcia, and Valencia.' From 1819 to 1821 it was a third time epidemic in the provinces of Andalusia, Murcia, and Catalonia. Since 1821 it has four times (1823, 1828, 1870, and 1878) been imported into Spain, but has not attained any great prevalence. In Portugal there was an epidemic in 1723, and another in 1856 (120 cases, 53 deaths—Hirsch), besides several other importations which did not result in any notable spread of the disease. In 1804 and 1821 the disease was carried from Barcelona to Majorca. In 1804 it was also imported into Leghorn from Cadiz. Cases have at different times been brought to several other European ports, but except as regards Brest (1856), Saint-Nazaire (1861), and Swansea (1865), no epidemic has followed. In Swansea, where the disease was clearly brought by a vessel from Cuba, some twenty-six cases, fourteen of which ended fatally, occurred among the residents of the locality,[1] but, it should be noted, exclusively among persons who either resided in, or were taken by their business relations into, the neighbourhood of the infected vessel.

The absolute limits of yellow fever distribution, according to present experience, are—for the Western Hemisphere, 34° 54′ south latitude (Montevideo) and 46° 50′ north (Quebec); for the Eastern Hemisphere, 8° 48′ south (Ascension) and 51° 37′ north (Swansea). In epidemic form, however, its northern limits have been 43° 4′ (Portsmouth, N. Hampshire), and 43° 34′ (Leghorn) in the Western and Eastern Hemispheres respectively.

Influence of Climate and Season.—Yellow fever is undoubtedly, to a large extent, controlled by climate and season. This is evident from its geographical distribution, and from the fact of its being most prevalent in the hot season of the year. Nevertheless, although heat is necessary for the development of an epidemic, the disease, when once well established, does sometimes persist in spite of cool weather, though it is invariably arrested by frost. But while it is always arrested by frost, *its cause* is not necessarily thereby destroyed, but may, like that of cholera, survive the winter, and give rise to a fresh epidemic on the return of hot weather. In the case of infected ships, too, it has on different occasions been observed that although the disease has died out as the higher latitudes have been reached, it has reappeared on the return of the same ships to warmer climates.

With regard to the influence of humidity, the evidence for different places

[1] See Sir George Buchanan's Report: *Eighth Report of the Medical Officer to the Privy Council*, 1865. The twenty-six cases include six doubtful ones.

is somewhat conflicting. According to Hirsch, a high degree of atmospheric moisture is generally favourable to yellow-fever prevalence. It has been stated by some observers, however, that in particular localities (the coast of Guiana and Guadeloupe) dry weather favours the prevalence of the disease.

Fatality.—The fatality of yellow fever varies largely in different epidemics. This is due to several causes, and in part, no doubt, to variations in the malignancy of the virus. It depends, however, also in part upon race, as will be seen immediately, and particularly upon the degree of resistance primarily acquired, as a result of acclimatisation, by those attacked. Among unacclimatised adults, the fatality ranges from 20 to 60, or even 80, per cent.

Influence of Race.—No race is entirely exempt from yellow fever, but there is no doubt that negroes are decidedly less susceptible than the white races. They are less liable both to attack and to death in the event of attack. It is further said that, broadly, the susceptibility of different people is inversely as the temperature of their native climate.

Influence of Sex.—Both the attacks and deaths are more numerous among males than females. This is, no doubt, largely owing to the greater exposure of males and to their habits—especially over-indulgence in alcohol. Whether, apart from these factors, males are actually more liable to attack and death by yellow fever than females is doubtful.

Influence of Age.—In localities in which the disease is endemic the majority of observed cases occur among persons in middle life, attacks among children and old persons being said to be relatively few. In its bearing upon the age incidence of the disease this circumstance is, however, perhaps somewhat misleading. During epidemics in endemic localities visitors, who have not yet become acclimatised, form a large proportion of the cases, and such visitors no doubt consist largely of adult males. And further, it appears that in localities in which yellow fever is not actually endemic, but occurs in occasional epidemics, large numbers of children are attacked—for, unlike the children in endemic areas, they are not acclimatised. Even in New Orleans, it seems that during epidemic periods children die in large numbers. In Table XIX.[1] Dr. Bemiss has shown the age distribution of 905 cases which occurred in that city during the epidemic of 1878. The age distribution of the deaths and the fatality are also shown.

TABLE XIX.

Age	Cases	Deaths	Per cent.
Under 5 years of age	206	26	12·67
From 5 to 10 years of age	233	20	8·61
From 10 to 20 years of age	183	9	4·9
From 20 to 40 years of age	232	39	16·7
From 40 to 60 years of age	47	6	12·7
From 60 to 80 years of age	4	2	50·0

Protection.—One attack of yellow fever usually confers immunity, though second attacks, apparently, sometimes occur. But apart from any recognised attack of the disease, there is no doubt that a large degree of immunity may be acquired by long residence in a yellow-fever country. With respect to this Dr. Sternberg remarks: 'It is a remarkable fact that the population of a large city like Havana, or Rio Janeiro, in which yellow fever has been endemic for a series of years, enjoys such a degree of immunity from the effects of the deadly poison that there is no interruption of business or pleasure at a time when strangers in the city are falling sick on every side.

[1] Taken from Dr. Sternberg's Report.

The development of an *epidemic* in these cities depends [? partly] upon the presence of susceptible strangers in sufficient number to furnish a series of cases considered large enough to justify the use of the word. . . . Under exceptional circumstances, however, epidemics are developed in these endemic foci of the disease, in which those who, by birth or long residence, were supposed to be acclimatised furnish a certain quota to the general mortality.' This is of interest as illustrating the fact that the infectivity of a given disease is not a fixed quantity, and that, therefore, a degree of immunity sufficient to be proof against infection during one epidemic may not prevent attack during another epidemic of the same disease. Hirsch, also, remarks that the 'peculiar immunity [of the negro] proves "insufficient" in severe epidemics of yellow fever.'

But it is certain that those habitually residing in yellow fever localities enjoy a large relative immunity, and the question arises whether such immunity is transmissible by heredity, or whether it is entirely acquired by each individual for himself. Many authorities have advocated the hereditary theory, and it seems possible that immunity is transmissible. On the other hand, it is more likely, as Dr. Sternberg says, that 'the creole child owes his immunity not to his parents, but to individual acclimatisation, and not unfrequently, to say the least, to a mild, unrecognised attack of yellow fever.' In support of this view Dr. Sternberg quotes Dr. Dowler to the effect that 'many creole children had, during the epidemic of 1853, a fever, a slight fever, yellow fever if you please, known as such rather by the co-existence of the epidemic than from any severe symptoms among these children, a slight fever never yet described, having generally but one paroxysm, lasting from six hours to one, two, or three days, scarcely ever requiring medication. That a few of these cases acquired an alarming violence, and even proved fatal, is most true, most deplorable.' Lastly, it is clear that the immunity due to 'acclimatisation' may be largely lost by residence outside the yellow fever zone, and this seems true also of the pronounced immunity of the (African born) negro race. Further, the immunity gained by residence in a particular yellow fever locality appears to be, to some extent at least, special to that locality. Thus, it has been pointed out by Humboldt and others that the natives of Vera Cruz, who remain exempt while at home, are liable to die of yellow fever if they migrate to Havana, or some other place in which yellow fever is endemic. According to Cornuel[1] also, if two bodies of troops stationed at different points in the Antilles exchange garrisons, yellow fever is apt to break out among both, though other troops who have remained in the same garrisons remain unaffected.

The most efficient and permanent immunity is, without doubt, that which results from a well-marked attack of yellow fever, though even this immunity has been alleged by some observers to be lost by long absence from endemic localities.

Cause and Mode of Dissemination.—Judging both from its clinical features and epidemiological behaviour, there are strong grounds for believing yellow fever to be a microbic disease, but as yet the particular micro-organism upon which it may be presumed to depend has apparently not been identified. Several observers, notably Dr. Domingos Freire, of Brazil, and Dr. Carmona y Valle, of Mexico, have described different micro-organisms which they regard as standing in causal relation to the disease; but Dr. Sternberg, who has specially studied the matter on behalf of the United States Government, and whose report has been already referred to, believes that he has been able to exclude, in a definite manner, each of these several micro-organisms.

[1] *Annal. Marit.* 1844, ii. 739.

It has already been seen that yellow fever is endemic only in certain localities, and the evidence seems to show conclusively that when it has occurred elsewhere its occurrence has been due to importation. It is true, no doubt, that in particular instances the disease, having been imported into previously uninfected places, has, notwithstanding that it has died down during the cold weather, survived the winter, to reappear, independently of any fresh importation, the following year. And, further, where the conditions are favourable, the importation of the disease may perhaps lead to the establishment of a fresh endemic centre; this has possibly occurred during the latter half of the present century in Brazil. But apart from such resuscitations and the establishment of new endemic foci—themselves, in the first instance, referable to importation—the occurrence of the disease elsewhere than in the Gulf of Mexico, the West Indies, and the Guinea Coast has practically, with few exceptions, been definitely traced to the arrival of persons or things from one or other of the localities last named. There is thus, so far, no evidence of the latter-day *de novo* origin of the disease, even in the senses indicated in the introductory section to this article.

And if the behaviour of the disease is studied in more detail, the same general result is brought out. 'An analysis of any yellow fever epidemic,' says Hirsch, 'shows certain groups of cases so arranged as to constitute separate foci of disease, sometimes in single houses, sometimes in blocks of houses, or, again, in streets or groups of streets; so that each new case of disease, as it occurs, may be traced to infection of the individual within any one such focus. That case may, in its turn, become the centre of a new focus. . . .' And in this way the extensions of yellow fever, once it has been introduced into non-endemic localities, may frequently be traced back to the original introduction.

There can be little doubt, then, that in a sense at least yellow fever is a communicable disease—a conclusion now accepted by almost all observers, including a board of American experts who investigated the epidemic of 1878, and who reported that 'the most frequent agency in the dissemination of yellow fever from place to place is found in yellow fever patients. . . .'

But strong as is the evidence upon which this conclusion is based, evidence equally strong is forthcoming to show that the communicability of yellow fever differs widely from that of small-pox, typhus, and other typically infectious maladies, and that in certain important respects yellow fever more resembles, in the matter of communicability, enteric fever and cholera. It is a matter of very general experience, for instance, that those in close attendance upon the sick do not specially contract the disease. 'It is well known to the people of the City of Mexico that a visit to the sea-coast city of Vera Cruz during the epidemic season is likely to result in an attack of yellow fever. It is also well established that those who fall sick with the disease after their return to the City of Mexico never communicate it to others who are closely associated with them as attendants, &c. . . . This is also the experience of the physicians in charge of hospitals—e.g. the Charity Hospital of New Orleans. So long as the hospital and its vicinity remain uninfected, cases do not originate in the hospital, although yellow-fever patients may be admitted to the wards with unacclimatised persons suffering with other diseases, and be cared for by susceptible attendants.' [1]

Similar testimony is given by Lawson for Sierra Leone. As regards the epidemic at Swansea in 1865, Sir George Buchanan points out in his report that 'persons [who had been] exposed to the fever-producing influences about the docks lay sick of yellow fever in various parts of the town . . .

[1] Sternberg, *op. cit.*, p. 57.

yet in no single instance out of all these did any person (whose business did not lead them to the infected neighbourhood of the docks) get yellow fever or any disease at all simulating it.'

Considering all the facts, then, it appears clear that the yellow fever patient does not usually at any rate directly infect others, but that he nevertheless gives off, in some way or other, probably with his discharges, the virus of the disease, and that this, if it should meet with suitable conditions, is capable of infecting the particular locality, and of thus, indirectly, giving rise to the disease in other persons. Outside the body the microphyte no doubt finds a habitat in the soil, and it has been observed by Thomas, Barton, and others, that breaking up the soil for the purpose of cutting canals, &c., in yellow fever countries has apparently given rise to outbreaks of the disease. It is also notable that the virus of yellow fever displays a special ability to establish itself in ships and dwellings. Epidemics on shipboard occupy a conspicuous place in the history of the disease; and that such epidemics have been due to the fact of the ships themselves becoming infected, and not to the direct transmission of the disease from case to case, is evident from the circumstances of the epidemics in question. Such epidemics, for instance, have frequently been limited to particular parts of the ship, as a single cabin, or one side of the vessel; and it has often been noticed that while the seamen have suffered, the officers and passengers have escaped. As regards the tenacity with which the disease clings to dwellings, Guyon, as a result of Lisbon experience, remarked of the infected house: '*whether there are still sick persons in it, or whether there are no longer any, that house will become a centre to reproduce the disease in the strangers who enter it;* and what is true of a whole house applies also to a part of it.'[1] Many observers, according to Hirsch, have reported to the same effect from North and South America, the West Indies, and Mexico.

It has been seen that the cause of yellow fever may be conveyed from place to place by the sick. But it may, too, be transported by fomites. In not a few instances an outbreak of the disease has followed the arrival of ships which, although coming from infected places, have apparently had no actual sickness on board, either at the time of their arrival or during the course of the voyage. Similarly, outbreaks in towns have followed the arrival of apparently healthy people from infected localities. A notable instance of this occurred at Madrid in 1878, when the persons first attacked were those who were closely associated with some soldiers who are said to have been themselves in good health, but who had recently come from Cuba, bringing with them their baggage.

There are, however, certain apparently conflicting facts with respect to the spread of yellow fever by the agency of infected persons and things which require a further hypothesis for their reconciliation. On the one hand, it is maintained that a single imported case may act as a focus for the spread of the disease, and that infected baggage from the yellow fever zone may also set up the disease elsewhere. On the other hand, it is said that 'even the most intimate kinds of contact, such as the healthy and the sick sleeping in one bed, the attendance of physicians and nurses upon the sick, the use of the uncleansed linen, clothes, or beds of yellow-fever patients, *post-mortem* examinations of their bodies, and the like, . . .'[2] have usually failed to spread the disease. In respect to this seeming paradox it has to be remembered that it is probably not every case of yellow fever that is capable of reproducing its kind. Moreover, it may be that the yellow fever microphyte is infective in certain phases and not in others, and particularly that, as has

[1] Hirsch, *op. cit.*, vol. i. p. 376. [2] *Ibid.*, *op. cit.*, vol. i. p. 372.

been often suggested with regard to cholera, it is not infective immediately after leaving the patient, but requires time to undergo some functional modification outside the human body. This is rendered probable from the experience that the 'first cases of local origin in an epidemic do not, as a rule, occur until some time has elapsed after the arrival of the infected ship or fomites or sick person responsible for the introduction of the "germ."'[1]

In addition to the conveyance of the yellow fever germ by persons and things, instances of the spread of the disease from one ship to another, or from the shore to the ship, or the ship to the shore, have often been brought forward as evidence of the carriage of the germ for considerable distances by the wind. But it is by no means clear that in the instances in question indirect human agency has been sufficiently excluded. For the present, therefore, the question must be left open, the probability, based upon the general behaviour of the disease, being against the transport of the virus to any distance by winds.

There is at present no evidence that yellow fever is spread by infected water or milk. The absence of evidence on these points, however, must not be taken as excluding the agencies in question, and, judging from the analogy of enteric fever and cholera, there is, as Parkes observes, '*à priori* probability that the cause is swallowed also in this case, and that it may possibly enter with the drinking-water.'

As regards the general conditions which favour the prevalence of yellow fever, heat no doubt occupies a foremost place. This, however, has already been considered. The influence of humidity has also been referred to. Another factor of importance is proximity to the sea. Yellow fever is essentially a disease of coast districts and the banks of great navigable rivers, particularly the former, and it seems only when the disease acquires a high epidemicity that it penetrates inland, its tendency to spread inland, for instance, being apparently much less than that of cholera. Altitude is also a factor which influences adversely yellow fever prevalence. In certain particular instances it is true that it has occurred in localities having considerable elevation. Thus, it has once or twice prevailed at Camp Jacob, in Guadeloupe (elevation 1,800 feet), and at Newcastle, in Jamaica (4,000 feet). But this is exceptional, and as a general rule it is a disease of the low-lying country. It does not appear, however, that it is especially prone to occur in malarious districts, as has so frequently been alleged—an allegation probably due to the frequency with which the severe forms of malarial disease have been mistaken for yellow fever. The latter disease has, in fact, often prevailed extensively in neighbourhoods relatively free from malaria; while, on the other hand, there are malarious districts in the yellow fever zone which are almost entirely free from yellow fever. Extremely strong arguments against the malarial nature of yellow fever are also found in the fact that yellow fever is almost entirely a disease of the towns, and that it occurs freely on board ship. On the whole, it would not appear that the disease is largely, if at all, influenced by the particular kind of soil. Insanitary conditions, however, seem to be of real importance. The 'places where the causes of the disease principally prevail are,' according to Bone, 'the vicinity of foul drains, the banks and channels of rivers which are dry at certain periods, the leeward openings of gullies, and crowded and ill-ventilated rooms, and ships with foul holds.' And there is a mass of evidence to the same effect from other observers.

Among the predisposing causes special to the individual, excessive fatigue, undue exposure to the sun, mental depression, and the debility following upon a debauch, are probably among the most important. As regards the

[1] Sternberg, *op. cit.*, p. 58.

last, Dr. Sternberg remarks that 'sailors who go on shore at an infected port for "a little spree" very commonly turn up in the hospital, or are taken sick after they come on board ship. . . .' Constipation and plethora have been also mentioned as predisposing conditions.

Period of Incubation.—This is usually short, ranging between twenty-four hours and four or five days. According to some observers, however, it may extend to fourteen days, or, exceptionally, even to five or six weeks. Such exceptionally long periods of incubation seem, from general experience, to be improbable, and it is more likely that they are to be explained by infection through the agency of fomites at a considerably later date than had been supposed.

P. 224 [illegible]

P. 354. Erysipelas from sewer air.

P 363 [illegible]

P 711 [illegible]

P 250 [illegible]

air borne infection

P. 701. Vault.

P. 320. Sewer air = Typhoid
322 "

The Evolution of Disease Germs in Water Supplies.

Journal [illegible] - 1[illegible]

Incubation Period, significance of p 264

p 265 reproduction of Bacteria Sexual?

p 269. Influence of temperature on Bacteria -

P. 247.

p 202 Immunity -